PREVALENCE AND ASSESSMENT OF EXCESSIVE DAYTIME SLEEPINESS IN DIABETIC AND OBESE PATIENTS

Dissertation submitted to

The Tamil Nadu Dr. M.G.R. Medical University, Chennai

In partial fulfillment for the award of the Degree of

MASTER OF PHARMACY

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Submitted by
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This is to certify that the dissertation work entitled **"PREVALENCE AND ASSESSMENT OF EXCESSSIVE DAY TIME SLEEPINESS IN DIABETIC AND OBESE PATIENTS"** was carried out by **JOSMY JOSE.** The work mentioned in the dissertation was carried out at the Department of Pharmacy Practice, KMCH College of Pharmacy Coimbatore – 641 048, under the guidance of **Mr. C.DHANDAPANI, M. Pharm.,** for the partial fulfillment for the Degree of **Master of Pharmacy** in **Pharmacy Practice** during the academic year 2011-2012 and is forwarded to The Tamil Nadu Dr. M.G.R. Medical University, Chennai.

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DECLARATION

I do hereby declare that the dissertation work entitled **"PREVALENCE AND ASSESSMENT OF EXCESSIVE DAYTIME SLEEPINESS IN DIABETIC AND OBESE PATIENTS"** submitted to the Tamil Nadu Dr. M.G.R. Medical University, Chennai in the partial fulfillment for the Degree of **Master of pharmacy** in **Pharmacy Practice,** was done under the guidance of **Mr C.DHANDAPANI, M pharm.** at the Department of Pharmacy Practice, KMCH College of Pharmacy, Coimbatore, during the academic year 2011-2012.

JOSMY JOSE

EVALUATION CERTIFICATE

This is to certify that the dissertation work entitled **"PREVALENCE AND ASESSMENT OF EXCESSIVE DAYTIME SLEEPINESS IN DIABETIC AND OBESE PATIENTS"** submitted by **JOSMY JOSE**, University **Reg No:26107283,** to The Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfillment for the Degree of **Master of Pharmacy** in **Pharmacy Practice** is a bonafide work carried out by the candidate at the Department of Pharmacy Practice, KMCH College of Pharmacy, Coimbatore and was evaluated by us during the academic year 2011-2012.

Examination Center: KMCH College of Pharmacy, Coimbatore

Date:

Internal Examiner **External Examiner**

Convener of Examination

ABBREVIATIONS

IGT	-	Impaired glucose tolerance
SWS	-	Slow wave sleep
T2DM	-	Type II Diabetes Mellitus
FBS	-	Fasting Blood Sugar
HbA_{1C}	-	Glycosylated Haemoglobin
REM	-	Rapid Eye Movement
T1DM	-	Type1 Diabetic mellitus
NREM	-	non rapid eye movement
GH	-	Growth hormone
OSA	-	Obstructive sleep apnea
ESS	-	Epworth Sleepiness Scale
PSQI	-	Pittsburgh Sleep quality Index
EDS	-	Excessive Daytime Sleepiness
HPA	-	Hypothalamic pituitary adrenal axis
OSAS	-	Obstructive Sleep apnea Syndrome
SNS	-	Sympathetic nervous system
BMI	-	Body mass index
CRP	-	C-Reactive Protein

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INTRODUCTION

Sleep is a complex neuro chemical process essential in humans for the maintenance of health. Sleep can be defined as a period of rest for the body and mind during which consciousness is in partial or complete abeyance and the bodily functions are partially suspended. Sleep restores bodily functions and it has a great role in cerebral changes to facilitate memory consolidation and cognitive functions. Sleep has a close relation with cardiovascular, immune and hormonal diurnal cycles.[1]

Excessive Daytime Sleepiness [EDS] is defined in International Classification of Sleep Disorders based on the behaviour of falling asleep, including difficulty maintaining alertness or wakefulness and unintentionally falling asleep.[2] Excessive daytime sleepiness is one of the most common sleep-related patient symptoms affecting about 20 percent of the world population. Persons with excessive daytime sleepiness are at risk of road traffic accidents and have poorer health than comparable adults. The most common causes of excessive daytime sleepiness are sleep deprivation, diabetic mellitus, obesity, obstructive sleep apnea and sedating medications. Other potential causes of excessive daytime sleepiness consist of certain medical and psychiatric conditions and sleep disorders, such as narcolepsy.[3]

Diabetes is a condition of endemic proportions with increasing prevalence. By 2030, it is expected to have an effect on almost 439 million people ie about 7.7% of total world population. Among Diabetes mellitus, Type 2 diabetes mellitus (T2DM) accounts for most cases of diabetes,ie about 85–95%. Among the factors considered responsible for this sharp increase in prevalence are increased geriatric population, unhealthy diet, overweight, obesity, and a sedentary lifestyle[4]. There is evidence that sleep impairment should also be considered

a T2DM risk factor because, in addition to some of the leading risk factors listed above, it seems to be independently associated with diabetes. Both the development and control of T2DM seem to be affected by sleep quality and duration[5]. Many researchers suggest that optimizing sleep duration and quality might improve the metabolic control of individuals with T2DM and Type 1 diabetes mellitus (T1DM). On the other hand, it should also be taken into account that, poor glycemic control in T2DM and T1DM individuals may favour the development of sleep disorders.[5-8] In addition to diabetes, overweight and obesity have been growing in prevalence at an alarming rate.[9] Sleep loss is also increasing sharply. Chaput et al.[10] reported that in 1960 the modal adult sleep duration was between 8.0 and 8.9 hrs, whereas in 2004 the number of sleeping hours of more than 30% of adults aged 30–60 years had dropped to less than 6 hours. This clearly shows that the interacting epidemics of diabetes, obesity and sleep impairment form a vicious circle.[11,12]

The increased prevalence of metabolic disorders in our society is aggravated by endemic voluntary postponement of bed time and by the current sedentary lifestyle, leading to epidemic proportions of obese people. Sleep disorders are commonly found in patients with type 2 diabetes.[13] Diabetes and chronic loss of sleep show the fact that, both affect millions and one depends on the other. Associated with the endemic condition of diabetes in our society, chronic sleep loss is increasingly common in industrialized societies and affects about 45% of all adults.[14] Sleep is a complex behavioural state that occupies one third of the human life span. Therefore proper sleep pattern is very much necessary to improve quality of life in diabetic and obese patients. However, there is increasing evidence that sleep also modulates the metabolic, endocrine and cardiovascular systems.[15] Sleep disturbances are common and can be detrimental to the health, mood, and quality of life of the people with diabetes.

A study revealed a marked reduction in glucose tolerance and insulin sensitivity after 8 nights of 5-hour bedtimes compared with 8-hour bedtimes.[16] Various mechanisms have been explored and suggested that there is a potential link between poor sleep patterns and negative health outcomes like impaired glucose regulation and subsequent increased risk of Type 2 DM.[5] Evidence from cross-sectional studies suggests that a diabetic condition may involve a reduction in sleep duration or an impairment of sleep quality.[17]

Excessive day time sleepiness [EDS] is characterized by persistent sleepiness and often a general lack of energy, even after having apparently adequate night time sleep. Sudden involuntary sleep onset and micro sleeps are common complications. Diabetes and obesity are the two main factors contributing to EDS.[18]

Sleep and type II diabetes

In the past, T2DM was considered a disease of older age groups. Today, it is well established that even with a higher incidence in older people, T2DM may occur at any age. This change is attributed to the spread of a 'Western modern society lifestyle', which includes higher food consumption, sedentary behaviour, and shorter sleep duration.[19]

Sleep deprivation has been shown to be a risk factor for impaired glucose tolerance (IGT), [17] weight gain, insulin resistance, and T2DM itself. [20-22] At the same time, evidence from some epidemiological studies indicates that not only short (6 hrs or less), but also long sleep duration (9 hrs or more) is associated with increased prevalence of IGT and T2DM [10,23] and with high fasting plasma glucose and high HbA1c levels.[24] A study conducted with 1139 men found that subjects reporting short sleep duration (5–6 hrs of sleep per night) were twice as likely to develop diabetes than those who slept between 6 hrs and 8 hrs, and those reporting long sleep duration (8 hrs) were more than three times as likely to develop diabetes

over a period of 15 years.[25] The reason why more sleep negatively impacts metabolism has recently become the subject of much speculation.

Evidence for a modulatory impact of sleep on many physiological functions, including metabolic regulation and endocrine release, has been reported more than four decades ago. Night time hormonal release and glucose control are dependent on the occurrence of specific sleep stages. [26-28]Human sleep is composed of rapid-eye-movement (REM) sleep and stages 1, 2 and 3 of non-REM (NREM) sleep. During the deeper stage of non-REM sleep, i.e. slow wave sleep (SWS) or stage NREM 3, brain glucose utilization and sympathetic nervous activity are decreased and parasympathetic nervous activity is increased, relative to both wake and REM sleep. SWS are also associated with robust elevations of growth hormone (GH) levels, while pituitary–adrenal activity is inhibited.[28] Hence SWS is likely to play a major role in total body glucose regulation. More recently, orexin neurons in the lateral hypothalamus have been identified as playing a central role in the maintenance of arousal as well as feeding behaviour, [29,30] suggesting an impact of sleep duration on appetite regulation.

Another cause of the daytime sleepiness in patients with diabetes is the increased levels of inflammatory cytokines.[31] Presently, these cytokines are accepted as mediators of sleepiness, and they are closely involved in the pathogenesis of Type 2 diabetes.[32]

Sleep and Obesity

Sleepiness is the inability to maintain alertness and manifests characteristic hypersomnic behaviours and decreased functional outcomes. Daytime sleepiness is a common outcome of sleep disturbances. Obesity is also biochemically associated with sleepiness. That is, adipose tissue produces somnolence inducing inflammatory cytokines such as tumour necrosis factor-alpha, interleukin-1 beta, and interleukin-6.[31]

The cause of symptomatic daytime sleepiness in the obese population clearly needs further evaluation. There are many features of obesity that drive various aspects of obesity generated disease and disability. Daytime sleepiness may be related to the extent of adiposity, the distribution of adiposity, or metabolic, inflammatory, physical, mental, or psychological disturbances experienced by obese subjects.[33]

The symptoms of daytime sleepiness is measured using a validated instrument, the Epworth Sleepiness Scale (ESS).and the sleep quality of the past month is measured by Pittsburgh Sleep Quality index[PSQI]. It is hypothesized that the extent of symptomatic daytime sleepiness, measured with the ESS would correlate with one or several of these broad areas and that disturbance in these areas would explain substantial variance of the ESS scores.[33]

The ESS represents a validated questionnaire containing eight items that measure a subject's expectation of dozing in eight hypothetical situations. A score greater then 10 is considered indicative of excessive daytime sleepiness and a PSQI score of six or more were considered poor sleepers.[34,35]

Other Factors Contributing To EDS[18]

- Insufficient quality or quantity of night time sleep.
- Misalignments of the body's circadian pacemaker with the environment
- An underlying sleep disorder, such as narcolepsy, sleep apnea, hypersomnia or restless legs syndrome.
- Disorders such as clinical depression or atypical depression and obesity
- Tumours, head trauma, anaemia, kidney failure, hypothyroidism or an injury to the central nervous system.
- Drug abuse.

- Genetic predisposition.

REVIEW OF LITERATURE

Abdulbari Bener et al., in their study findings observed that disturbed sleep was more prevalent in the diabetic population. Also, excessive daytime sleepiness was observed more in diabetic patients, especially in women. Sleep loss varied significantly by gender in diabetic patients. A significant difference was observed in ESS scores between both genders. Obesity was more common among diabetic women with poor sleep than men, while physical activity was significantly less in women compared to men.[36]

Adriana et al., in their study in Romanian obese Type 2 Diabetic patients revealed that EDS is highly prevalent in Romanian Patients with Type 2 Diabetes and should be systematically screened for, especially among obese individuals with higher waist circumference and poor diabetes control.[37]

O. Resta et al., in their study clearly shows that severe obesity, even in the absence of OSAS, is associated with sleep-related disorders and EDS. All these alterations may be partly responsible for a lower quality of life, a higher prevalence of medical complications, an increased risk of occupational injury, and both social and family problems characterizing obese patients, independently of the presence of OSAS. Persons with excessive daytime sleepiness are at risk of motor vehicle and work-related incidents, and have poorer health than comparable adults. The evaluation and management of excessive daytime sleepiness is based on the identification and treatment of underlying conditions (particularly obstructive sleep apnea), and the appropriate use of activating medications.[38]

Kevin Ruggles et al., in their study suggest that, EDS is a common condition which results in a significant public health problem. Patients suffering from EDS are at increased risk for automobile accidents and work related injury. There is also evidence that sleep disorder are generally under diagnosed and under treated.[39]

John B. Dixon et al., in their study showed the presence of excessive daytime sleepiness, as measured by the ESS, was common in obese subjects but was not related to the presence or severity of OSA or other polysomnography findings in a selected group. It was related to older age, male gender, smoking, type 2 diabetes, symptoms of depression, and poor quality of life. It was also strongly associated with symptoms of disturbed nocturnal sleep.[33]

E. O. Bixler et al., in their study indicated that when diagnosing a case with a complaint of EDS, sleep disturbance (*e.g.* due to sleep apnea) should not be considered the only cause. It appears that EDS is more strongly associated with mood factors (*e.g.* depression) as well as metabolic factors (obesity and/or diabetes), *i.e.* the metabolic syndrome. EDS appears to be more prevalent in the very young, suggesting unmet sleep needs and/or depression. EDS is also more prevalent in the very old, most likely associated with increasing medical illnesses and health issues. Their findings indicated that patients with a complaint of EDS should be adequately assessed for depression, obesity, and/or diabetes both in the presence or absence of sleep apnea and then treated appropriately.[40]

Anna-Kaisa Renko et al., in their study found out the relation of snoring, sleep apnea and daytime sleepiness as independent indices of obesity related to Type II diabetes (T2DM).Their study supports the earlier findings showing that sleep apnea is fairly common

among subjects aged 61–63 years, and that male gender and BMI are independently associated with sleep apnea. Diabetic subjects suffer more often from habitual snoring than normoglycemic ones, and it seems that T2D, but not IGR, is independently associated with snoring. Daytime sleepiness seemed to be linked with depression but not with using the sleep medication, IGR and T2D. Their study also supports the previous findings showing that sleep apnea, habitual snoring and daytime sleepiness are connected with anthropometric measures.[41]

Eileen R. Chasens et al., in their study explored the association between daytime sleepiness on physical activity and functional outcomes in veterans with T2DM. It is suggested that daytime sleepiness is highly prevalent in male veterans with T2DM and that it is associated with decreased physical activity and poor functional outcome. They concluded that daytime sleepiness may impede physical activity among veterans with diabetes, confounding self-management.[42]

Tze Pin Ng et al., in their study affirmed the high prevalence of EDS in an Asian multi-ethnic population. Daytime sleepiness is associated with a wide multiplicity of risk factors in the community, including sleep behaviour, breathing, work and medically related factors, representing identifiable risk groups in the general population for targeted intervention. Many of these risk factors are highly prevalent in the population, and contribute substantially to the public health significance of EDS in the community.[43]

Mark.T.U.Barone et al., in their study suggested that sleep debt, OSA, obesity and T2DM are part of a vicious circle, where one influences, provokes, or impairs the others. Sleep exerts marked modulatory effects on glucose metabolism by influencing the balance

and levels of hormones, including leptin, ghrelin, insulin and cortisol, in addition to the activity of the SNS. Sleep loss and sleep disorders, e.g., OSA, can be understood as stressors, triggering SNS activation, secretion of stress hormones, and inflammatory responses. These physiological defense mechanisms alter glucose tolerance and sensitivity to insulin and leptin, impairing appetite regulation. Therefore, when chronically activated, these stress responses favour the development of obesity and T2DM. In addition, shorter sleepers have more opportunity for food consumption and tend to be more fatigued and less active during the day, which favour obesity. On the other hand, obesity itself, especially when central, leads to OSA, to inflammatory processes, and to the development of T2DM. Thus, they concluded that it is difficult to isolate a cause and an effect from this neuro-endocrine-metabolic misbalance, since sleep characteristics (disorders or duration) may impact neurological and endocrine systems to promote obesity and T2DM, while obesity and T2DM may impact sleep as well. Therefore, while obesity and T2DM are favoured and aggravated by short sleep duration or sleep disorders, sleep may be impaired by these two widespread metabolic conditions.[44]

Dev Banerjee et al., in their study recognised that Excessive Day time Sleepiness (EDS) has detrimental consequences such as road traffic accidents, impaired psychological functioning and reduced work performance. EDS can result from multiple causes such as sleep deprivation, sleep fragmentation, neurological, psychiatric and circadian rhythm disorders. Treating the underlying cause of EDS remains the mainstay of therapy but in those who continue to be excessively sleepy, further treatment may be warranted. Recently, the advent of modafinil has broadened the range of therapeutic options. Modafinil has a safer side effect profile. Traditionally the amphetamine derivatives, methylphenidate and pemoline psychostimulants were the commonest form of therapy for EDS particularly in conditions such as narcolepsy. [4]

OVERVIEW

DEFINITION

Excessive Daytime Sleepiness [EDS] is defined in International Classification of Sleep Disorders based on the behaviour of falling asleep, including difficulty maintaining alertness or wakefulness and unintentionally falling asleep.[2]

AETIOLOGY

Excessive daytime sleepiness occur due to hypersomnias

- Primary hypersomnia of central origin
- Secondary hypersomnia.

Narcolepsy is the most common of primary hypersomnia reported. Less common are the primary hypersomnia of Idiopathic origin and other rare primary hypersomnias like Kleine-Levin syndrome and menstrual hypersomnia.

Secondary hypersomnias are caused due to Sleep disorders. Sleep related breathing disorders include excessive daytime sleepiness secondary to obstructive sleep apnea. Behavioural sleep deprivation is common in adolescents and shift workers. Other sleep disorders include circadian rhythm sleep disorders, sleep-related movement disorders. Medical or psychiatric conditions also lead to secondary hypersomnias. Medication effects include prescription, non prescription, and drugs of abuse. Psychiatric conditions include depression. Medical conditions like head trauma, stroke, cancer, inflammatory conditions like encephalitis, neurodegenerative conditions can also lead to secondary hypersomnias.

Excessive sleepiness caused by a primary hypersomnia of central origin (e.g., narcolepsy, idiopathic hypersomnia) is less common.[46]

SLEEP DEPRIVATION

Sleep deprivation is probably the most common cause of excessive daytime sleepiness. Symptoms can occur in healthy persons even after mild sleep restriction. Most types of chronic insomnia including primary insomnia, psychopathological insomnia, and paradoxical insomnia are associated with daytime hyper arousal rather than excessive daytime Sleepiness. The presence of excessive daytime sleepiness in a patient with insomnia suggests co- morbidity such as a sleep-related breathing disorder or a mood disorder.[47]

MEDICATION CLASSES COMMONLY ASSOCIATED WITH DAYTIME SLEEPINESS [48]

Alpha-adrenergic blocking agents

Anticonvulsants (e.g., hydantoins,succinimides)

Antidepressants (monoamine oxidase inhibitors, tricyclics, selective serotonin reuptake inhibitors)

Antidiarrhoea agents

Antiemetics

Antihistamines

Antimuscarinics and antispasmodics

Antiparkinsonian agents

Antipsychotics

Antitussives

Barbiturates

Benzodiazepines, other γ-aminobutyric acid affecting agents, and other anxiolytics

Beta-adrenergic blocking agents

Genitourinary smooth muscle relaxants

Opiate agonists and partial opiate agonists

Skeletal muscle relaxants

PATHOPHYSIOLOGY

Although the pathophysiological pathways linking sleep to diabetes are still under study. It is not as simple as a linear cascade, but rather it is a complex and multifactorial neuro-endocrine-metabolic network. This complex, interactive system is divided into its best-known components below.[11,49]

1. Sympathetic contribution

Activation of the sympathetic nervous system (SNS) is one of the best understood consequences of stressful situations, such as OSA and other sleep impairment, including sleep curtailment.[20,50,51] Among the pathways through which SNS activation is likely to predispose to obesity, glucose impairment, metabolic syndrome and T2DM [52,53], the two most well known are the inhibition of leptin secretion[54] and the stimulation of the hypothalamic–pituitary–adrenal axis (HPA), resulting in excessive cortisol secretion, which impairs glucose homeostasis.[51,55]

2. HPA axis contribution

SNS, activated by sleep impairment, stimulates the HPA axis. Cortisol, a glucocorticoid secreted as a result of HPA activity, has effects throughout the body, some of them tightly correlated to metabolism in peripheral tissues. Exposure to excessive glucocorticoid levels leads to insulin resistance, weight gain and metabolic syndrome, by increasing glucose output and lipogenesis, decreasing glucose utilization and inhibiting lipid mobilization in the presence of insulin, especially from the visceral adipose tissue.[23,55-57]

Sleep disorders affect both the SNS and the HPA axis, negatively impacting carbohydrate metabolism, favouring the development of glucose intolerance, which might lead to T2DM. On the other hand, it is interesting to note that HPA hyperactivity is a known cause of sleep impairments such as insomnia, sleep fragmentation, shortened sleep time.[23, 55, 58]

3. Appetite regulation contribution

Sleep disorders impact the regulation of appetite, leading to decreased satiety and increased caloric intake, which strongly contribute to the development of obesity. Obesity is one of the main components of the metabolic syndrome and is a step toward T2DM and OSA.[12] There are several hormones and neuropeptides involved in appetite regulation such as cholecystokinin, glucagon-like peptide-1,and peptide YY3-36, among others, but the more important hormones that affect sleep impairment are leptin, insulin and ghrelin. Leptin is primarily produced by adipocytes, and its level in the blood is proportional to fat mass. Insulin is secreted by pancreatic beta-cells acutely in response to food intake. Those hormones convey anorexigenic information to the hypothalamus, suppressing appetite and affecting energy expenditure. The administration of exogenous leptin fails to reduce adiposity in most cases of human obesity. The reason for this is a state of leptin resistance, which explains the high circulating leptin levels observed. [59] The possible mechanisms of leptin resistance include the limitation of its transport into the brain, inhibition of leptin signaling pathways in leptin-responsive hypothalamic neurons, high serum levels of leptin-interacting proteins, or genetic mutation in its receptor.[59,60,61] As expected because of leptin resistance in overweight, its serum level has been shown to correlate positively with BMI, insulin, insulin/glucose ratio, AHI, and oxygen desaturation time. [62]

In contrast with the hunger- and appetite-suppressing signals of leptin and insulin, ghrelin conveys an appétite stimulating message to the hypothalamus.[63] Ghrelin is primarily secreted by the stomach and rapidly suppressed by food intakes. [59,64] Studies have found that short sleep duration is associated with decreased levels of leptin and increased levels of ghrelin. [11,65]

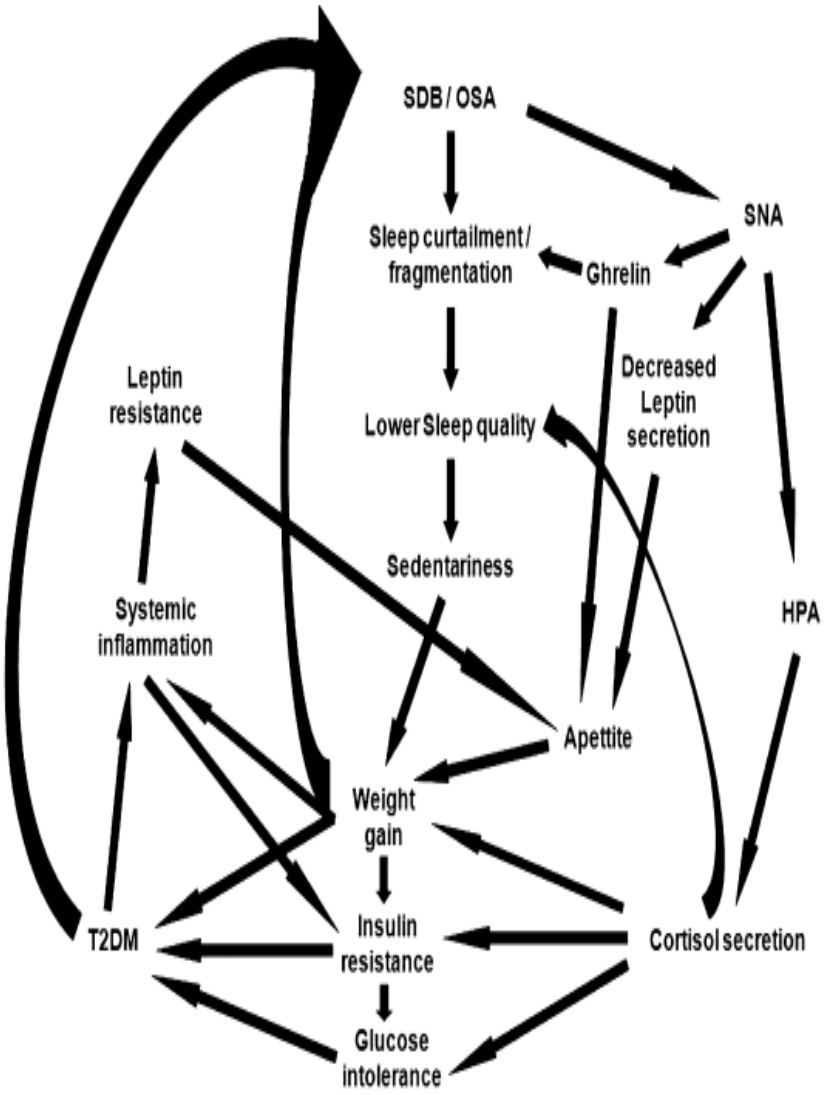
Therefore, impairment of appetite regulation might be a consequence of short sleep duration or due to leptin and insulin resistance (the hallmarks of obesity and T2DM) and be exacerbated by OSA. To explore the association between the sleep–wake cycle, appetite and metabolism, it is necessary to consider the role of the neuropeptides called orexins, orhypocretins. Orexin A and orexin B are excitatory neuropeptides found in the lateral hypothalamus and perifornical area. They are both stimulated by ghrelin, promote wakefulness, and increase appetite and SNS activity. Moreover, besides the down regulation of satiety, the promotion of short sleep time, and the stimulation of the HPA axis by increased SNS activation, ghrelin promotes adipogenesis and decreases energy expenditure, fat catabolism and lipolysis. Therefore, this might be one of the main reasons why individuals with short sleep duration are prone to gain weight and increase the risk of developing OSA and T2DM.[63,64]

3. Contributions of inflammatory processes

Obesity and T2DM are known to be associated with chronic systemic inflammation[66]. C - reactive protein was identified as one of the major serum leptin interacting proteins, and it seems to be one of the main agents responsible for leptin resistance.[61] Human CRP directly inhibits the binding of leptin to its receptors and blocks its ability to signal in cultured cells. In addition to sleep restriction, Cytokines may either promote or inhibit sleep through their interactions with different brain regions. Some of the sleep alterations that cytokines may

induce could be mediated by changes in nitric oxide synthesis, effects on neuro hormonal systems such as growth hormone releasing hormone, and activation of the HPA axis. Therefore, since obesity, T2DM, sleep disorders and sleep restriction are associated with increased inflammatory response, it is concluded that the pro-inflammatory effects of one disorder influence the expression of the other disorder.[67]

Pathway composing the vicious circle of T2DM and Sleep



TREATMENT

Addressing the underlying cause is the mainstay of treatment of excessive daytime sleepiness. **Modafinil (Provigil)** is considered to be the first-line activating agent for the treatment of excessive daytime sleepiness. It is indicated for the treatment of persistent sleepiness associated with OSA in patients already being treated with CPAP, and for the treatment of daytime sleepiness in patients with shift work disorder. Modafinil is pharmacologically distinct from and has a much lower potential for abuse (Schedule IV) than the amphetamines, and has a generally benign side effect profile. Other medications that must be used with caution to induce alertness in somnolent patients include the amphetamines (dextroamphetamine [Dexedrine], methylphenidate [Ritalin]) and pemoline.[68-70]

METHODOLOGY

Objective:

To study the prevalence of Excessive Day time Sleepiness [EDS] in diabetes and obese patients by assessing the quality and quantity of sleep by Epworth[ESS] and Pittsburgh Sleep Quality Index [PSQI] Scale and also to provide patient education to improve quality and quantity of sleep.

Study Design:

It is a prospective observational study.

Study Site:

The study was conducted in the department of Diabetology, Kovai Medical Center and Hospital, a multispecialty hospital in Coimbatore, Tamilnadu.

Study Period:

The study was conducted over a period of five months from September2011 to January 2012.

Study Population:

Both male and female patients diagnosed with Type II Diabetes Mellitus & Obesity within the age group of 20-65 were included in the study.

Sources of Data:

The data was collected through direct patient interview and also from various sources such as patient's case sheet, treatment chart and laboratory reports.

Study Procedure:

In this prospective observational study, both male and female patients who are diagnosed with Type II Diabetes mellitus, overweight and obese patients within the age group of 20-65 are included. The patients who do not wish to complete the questionnaire and those diagnosed with obstructive sleep apnea are excluded. Hospital Ethical Committee approval was obtained. All the patients are taken directly from the doctor's consultation room. The laboratory values and medications are noted from the patient's chart.

Data are collected and assessed by providing two questionnaires namely Epworth Sleepiness Scale [ESS] and Pittsburgh Sleep Quality Index [PSQI]. The former assess the daytime somnolence in patients and the latter assess the sleep quality during the past month.

The Epworth sleepiness scale is a brief, valid, reliable measure used to assess the likelihood that an individual will fall asleep in a series of situations such as watching TV, sitting and reading, sitting inactive in a public place like a theatre or meeting, travel in a car as a passenger for an hour without break, lying down to rest in the afternoon when circumstances permit, sitting and talking to someone, sitting quietly after lunch without alcohol and travel in a car while stopped for a few minutes in the traffic. The scoring of the answers is 0-3, with 0 being "would never doze", 1 for "slight chance of dozing", 2 for "moderate chance of dozing" and 3 for high chance of dozing". A score lower than 10 distinguishes as getting enough sleep, 10-12 comes in the borderline and greater than 12 as very sleepy and they should seek medical advice.

Pittsburgh sleep quality index (PSQI) was used to check the sleep habits of diabetic patient during the past month. The situations in the questionnaire were classified as during the past month how often had they taken medicine to help them sleep, wake up in the middle of the night or early morning, cannot get to sleep within 30 minutes, have to get up to use the bathroom, had bad dreams, have pain. For each of the questions of the questionnaire, response was classified as "never", "once a week" and "more than once a week". Individuals with a score of six or more were considered poor sleepers. Patient counselling to improve the quality and quantity of sleep were also given.

Data Analysis

Chi-square test will be applied to find out statistical significance of BMI, Physical activity, Age group factors on prevalence of excessive day time sleepiness in diabetic and obese patients.. Values of $P < 0.05$ were considered statistically significant.

Table: 1 GENDER WISE DISTRIBUTION OF STUDY POPULATION

S.No	Gender	N=160	Percentage
1	Male	88	55
2	Female	72	45

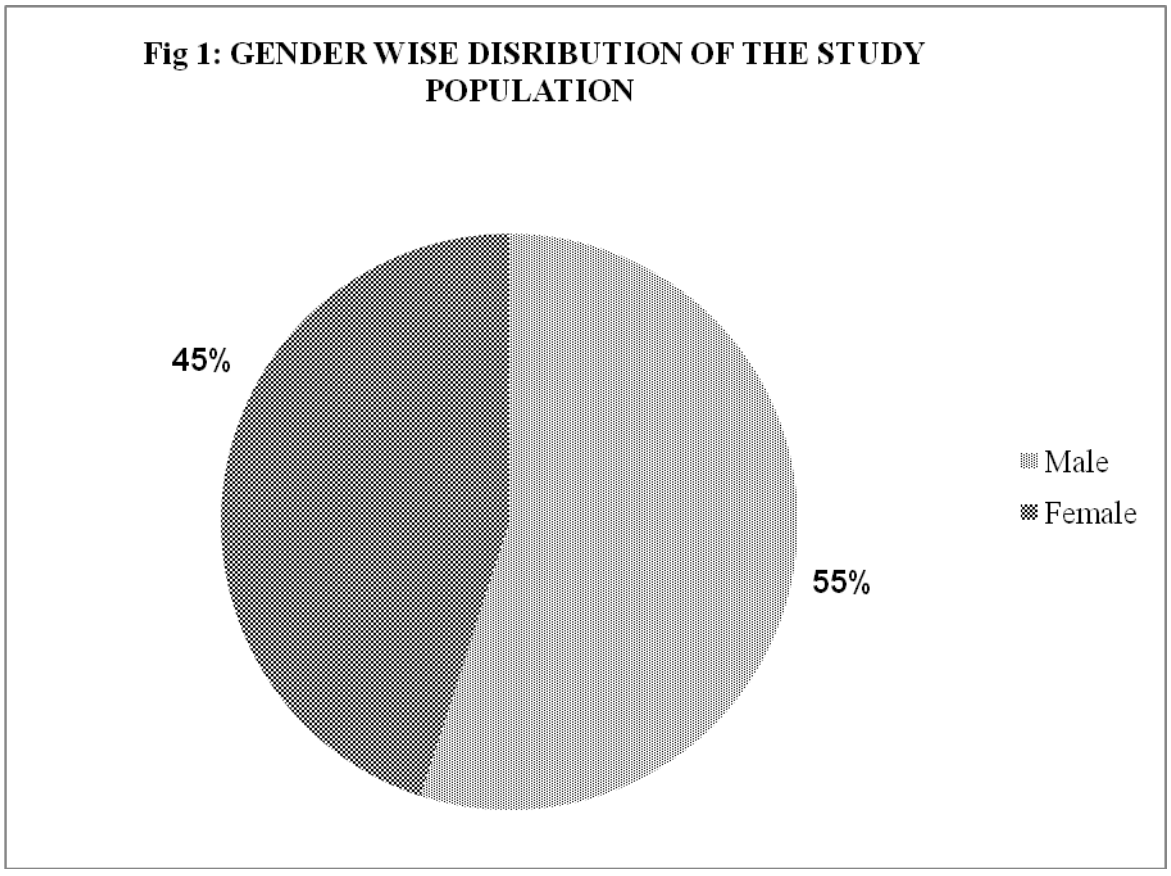


Table: 2 AGE WISE DISTRIBUTION OF STUDY POPULATION

S.No	Age Group	N=160	Percentage
1	25-35	8	5
2	36-45	36	22.5
3	46-55	54	33.7
4	56-65	62	38.7

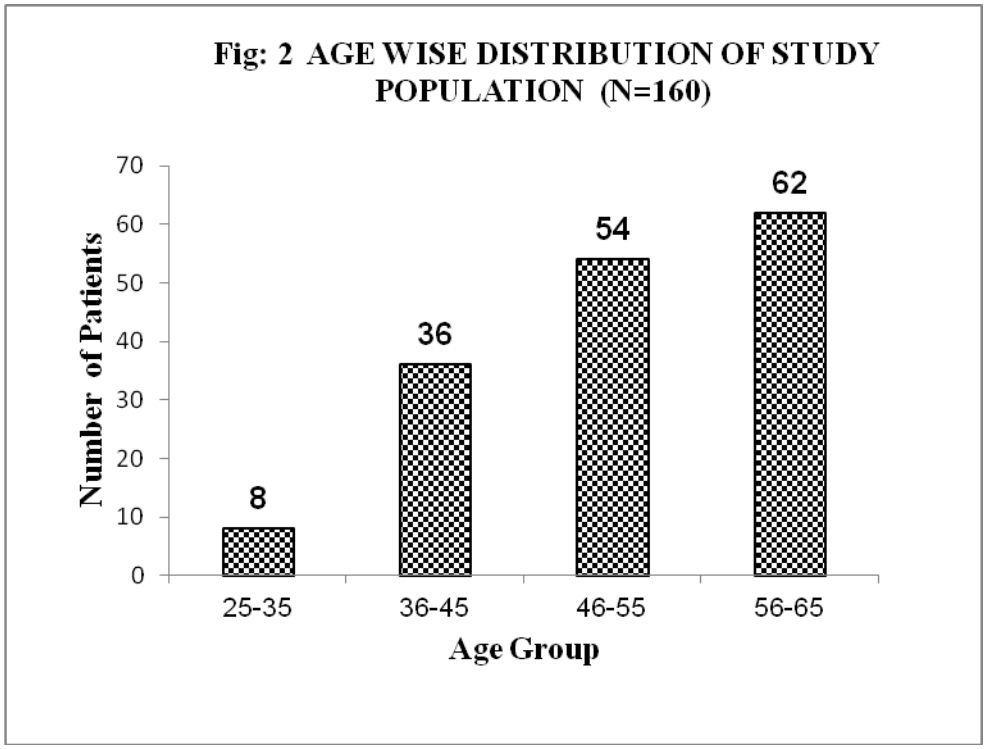


Table: 3 EDUCATIONAL QUALIFICATION AMONG STUDY POPULATION (N=160)

S.No	Educational Qualification	Male(%)	Female(%)
1	illiterate	12.5	33.3
2	1-10	25	20.5
3	10-12	37.5	23.1
4	≥Degree	25	23.1

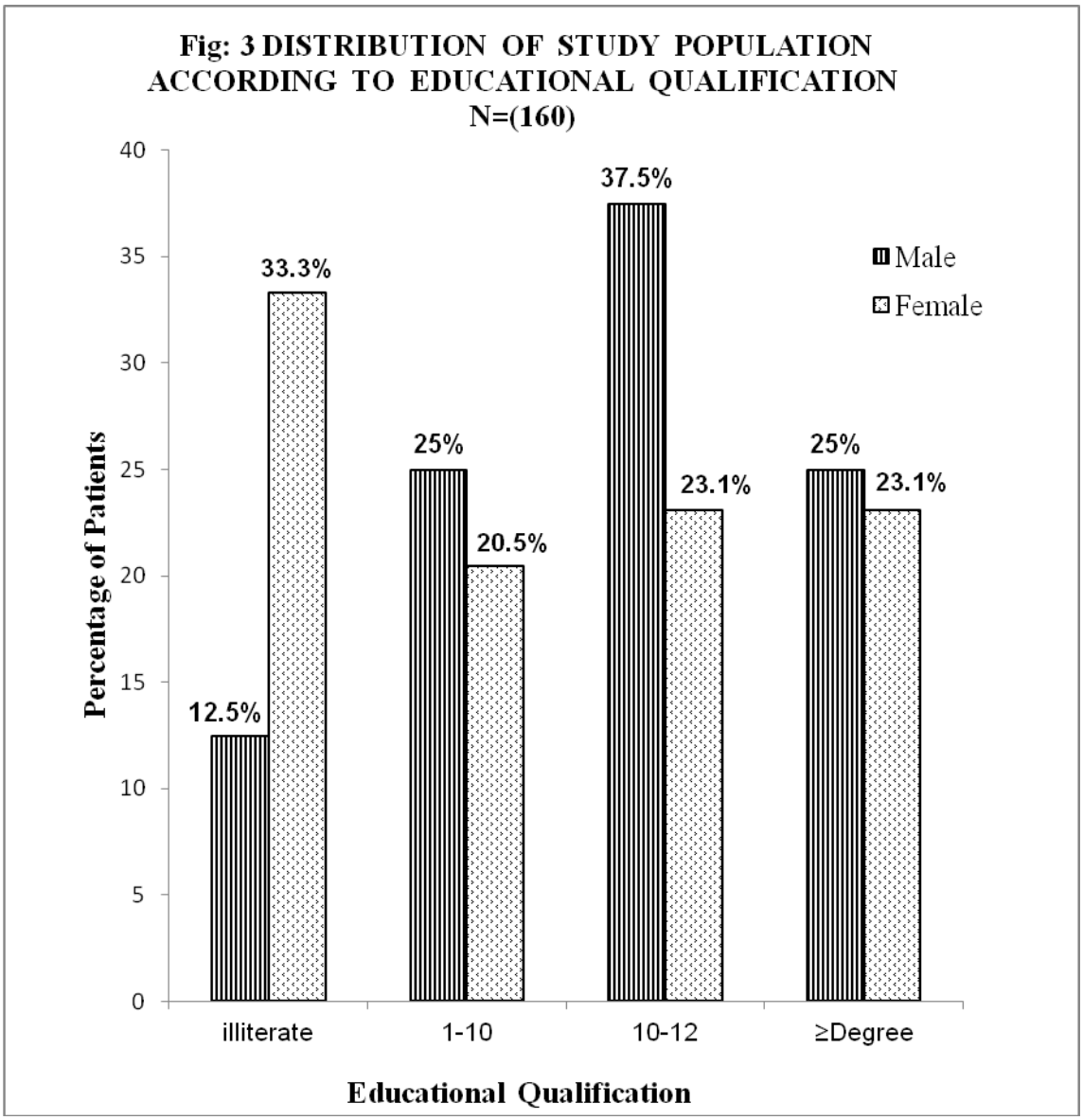


Table: 4 MONTHLY INCOME AMONG STUDY POPULATION (N=160)

S.No	Monthly income	Percentage of Patients
1	<5000	46.8
2	5000-15,000	36.7
3	>15,000	16.5

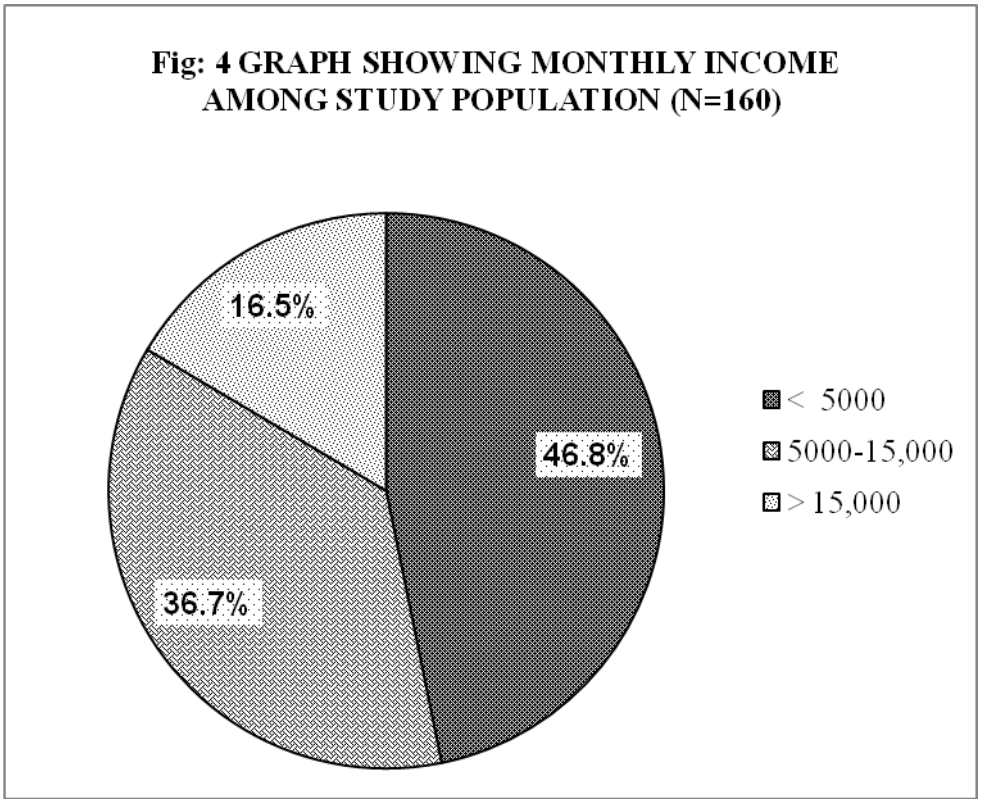


Table: 5 GENDER WISE PREVALENCE OF EDS

Sl No.	Gender	With EDS	Without EDS
1	Male	40	48
2	Female	39	33

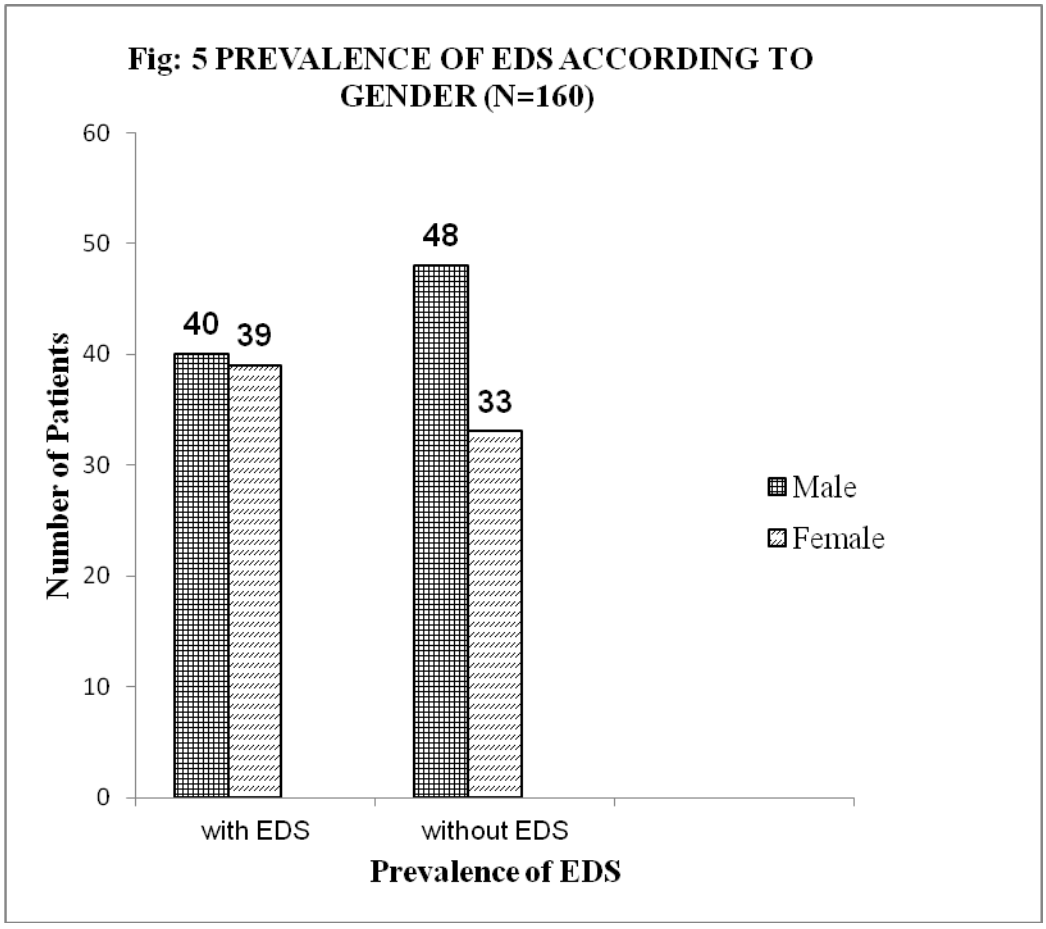
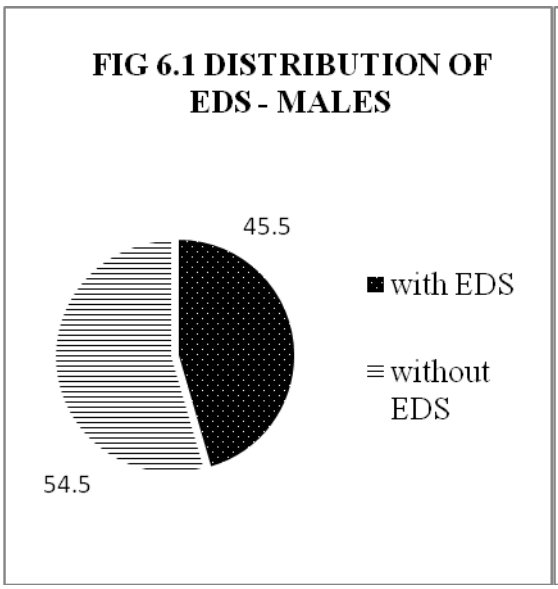


Table: 6 GENDER WISE DISTRIBUTION OF PREVALENCE OF EDS AMONG STUDY POPULATION

S.No	Gender	No. of patients	Percentage of Patients
1	Male	88	45.4
2	Female	72	54.2



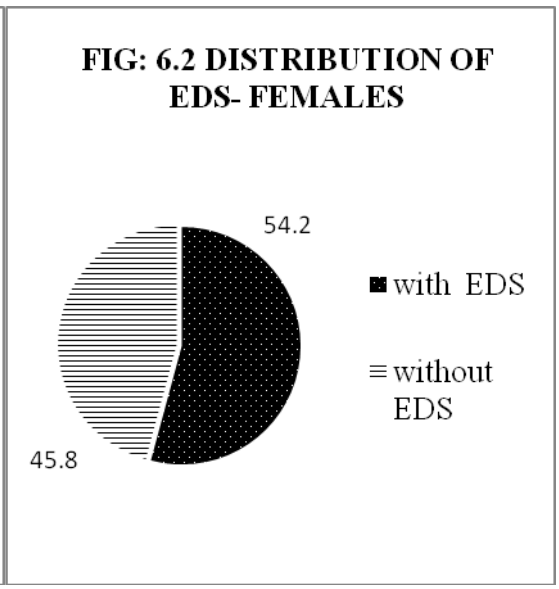


Table: 7 AGE WISE DISTRIBUTION OF PREVALENCE OF EDS

S.No	Age Group	Number of Patients	Percentage of patients	P value
1	25-35	4	5.1	0.300
2	36-45	14	17.7	
3	46-55	25	31.6	
4	56-65	36	45.6	

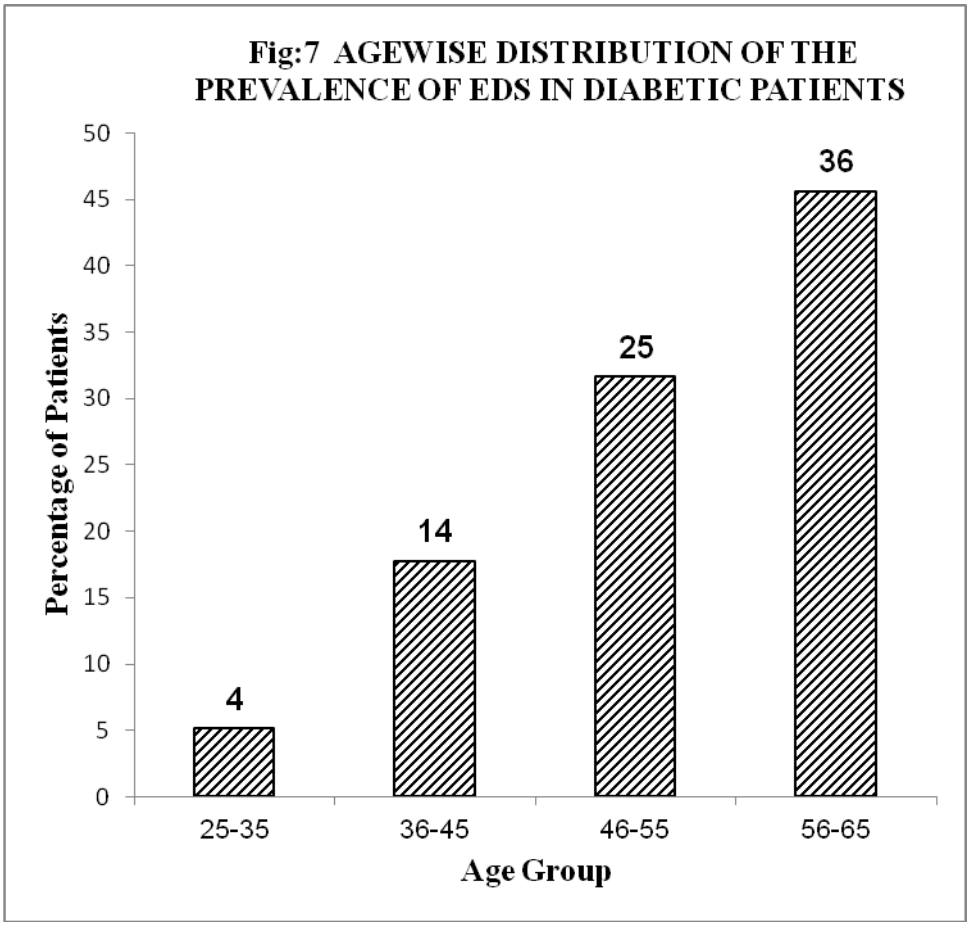


Table: 8 PREVALENCE OF EDS IN REGARD TO BMI OF THE SUBJECTS (N=79)

S.No	BMI	With EDS(%)	Without EDS(%)	P value
1	18-24.9	31.2	68.8	0.000
2	25-29.9	62.3	37.7	
3	≥ 30	84.2	15.8	

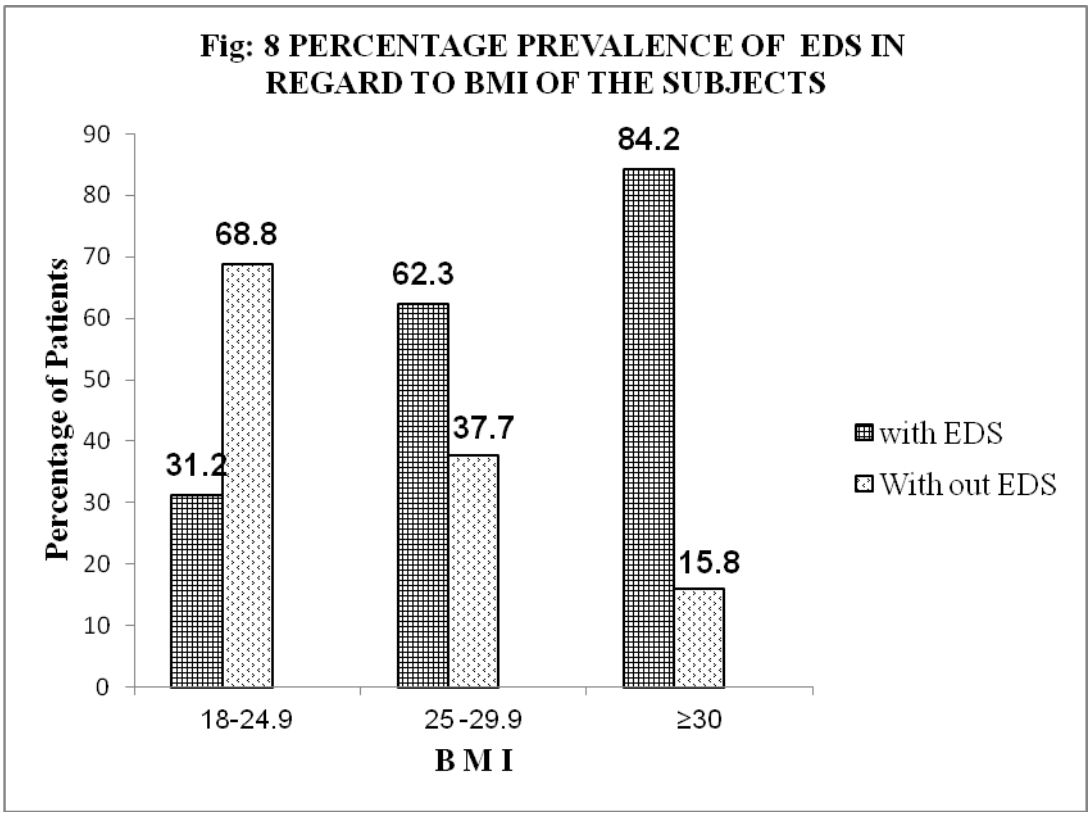


Table: 9 BMI OF BOTH MALE AND FEMALE PATIENTS WITH EDS

S.No	BMI	Male(%)	Female(%)
1	Normal(18- 24.9)	37.5	25.6
2	Overweight(25-29.9)	40	56.4
3	Obese(≥30)	22.5	17.9

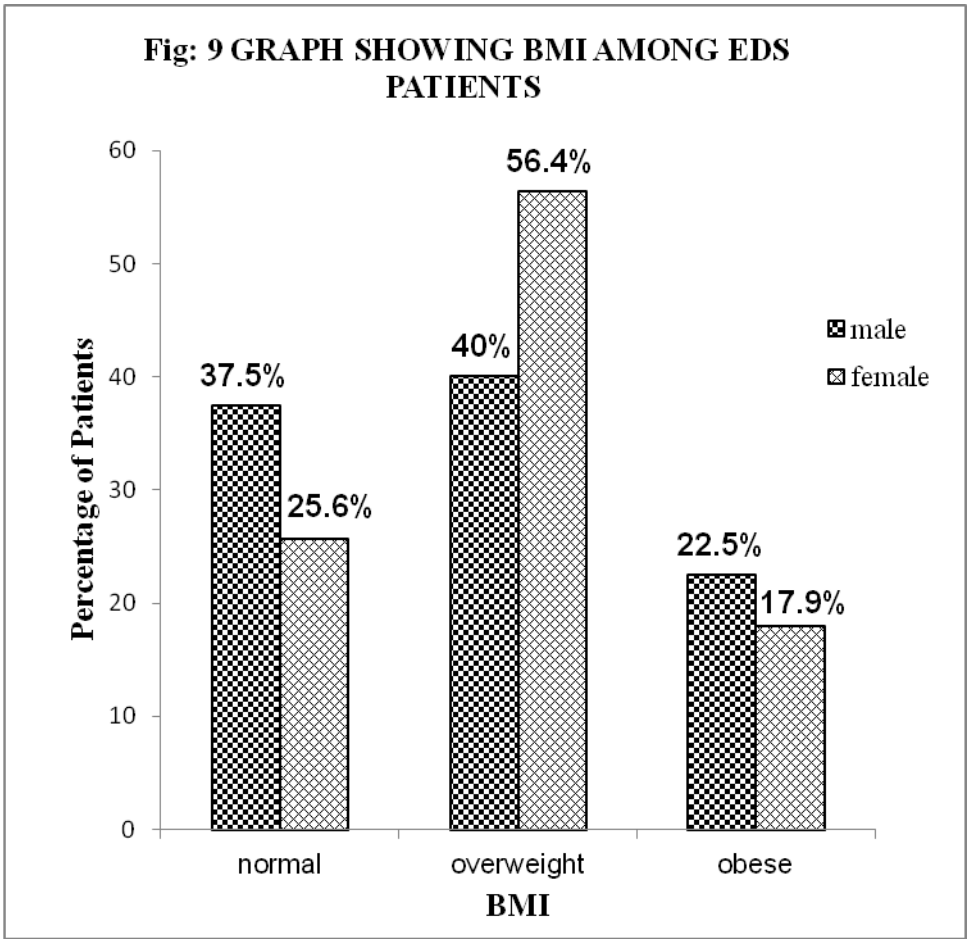


Table: 10 PREVALENCE OF EDS IN REGARD TO PHYSICAL ACTIVITY OF SUBJECTS

S.No	Physical Activity	With EDS(%)	Without EDS(%)	P Value
1	Moderate	26	74	0.000
2	Sedentary	59.5	40.5	

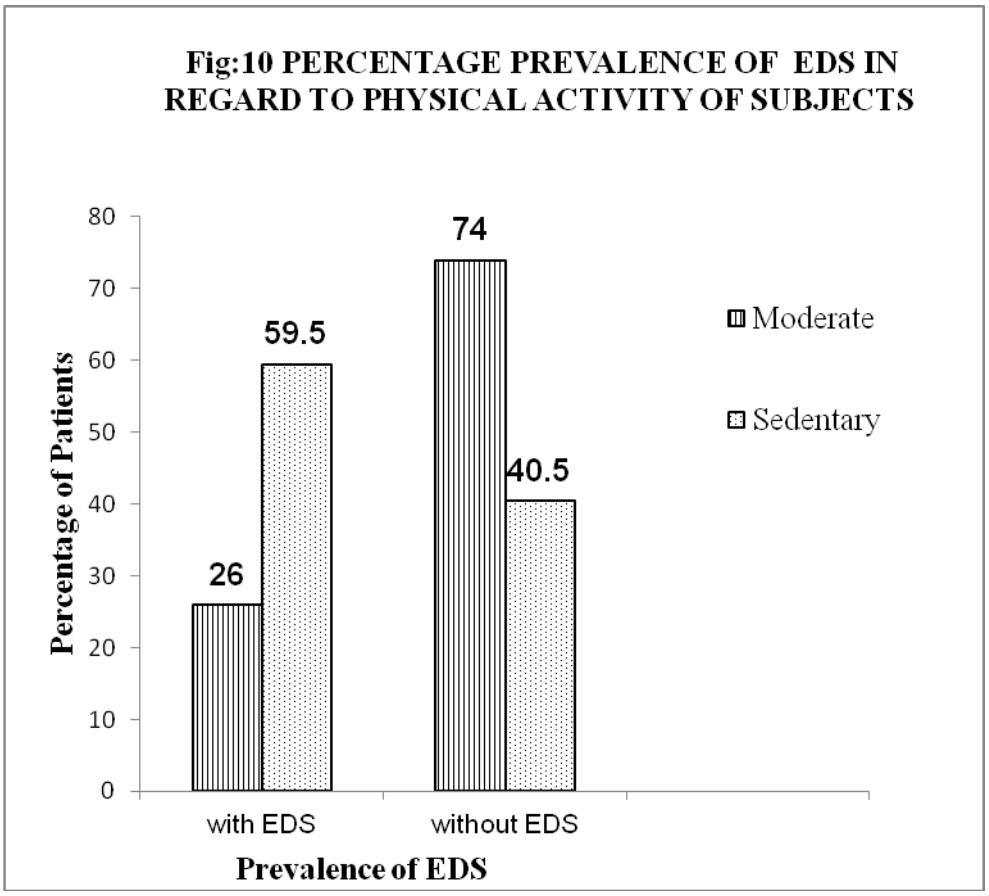


Table: 11 PHYSICAL ACTIVITY OF PATIENTS WITH EDS

S.No	Physical Activity	Male(%)	Female(%)
1	Sedentary	77.5	89.7
2	Moderate	22.5	10.3

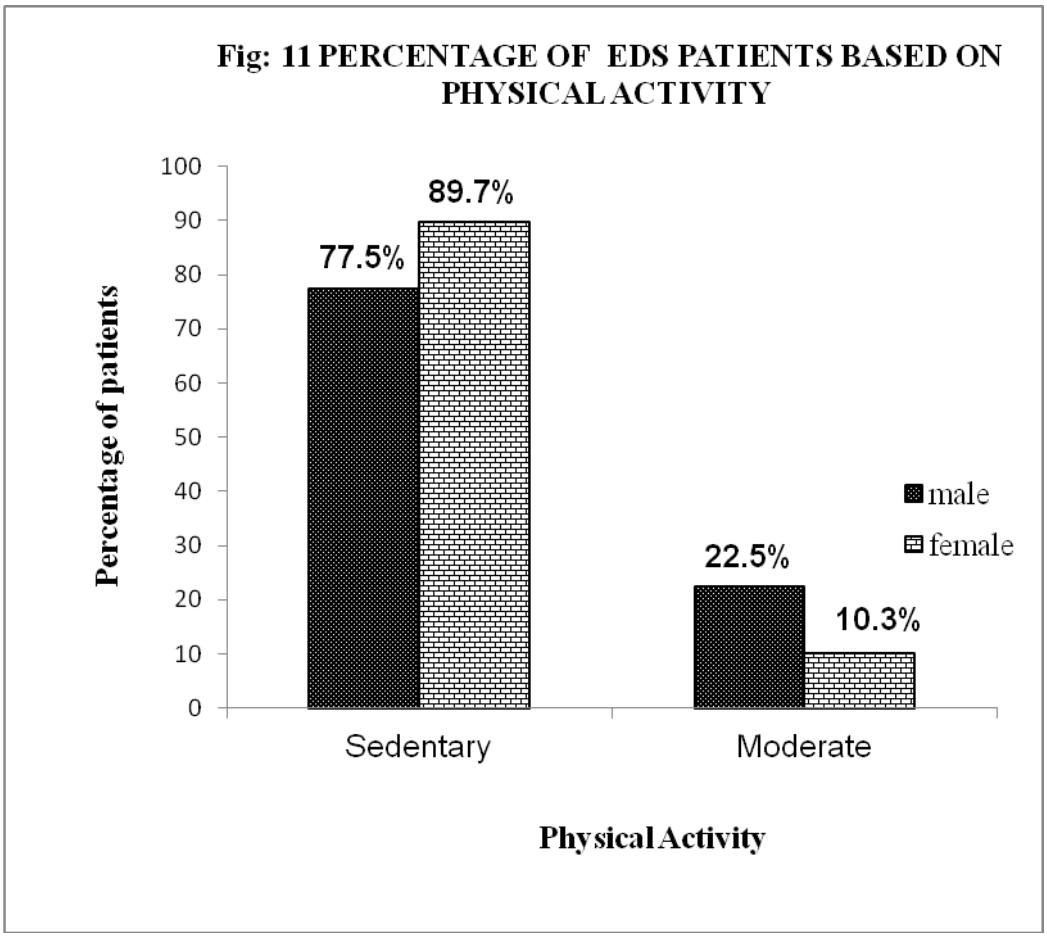


Table: 12 PREVALENCE OF EDS IN REGARD TO DURATION OF DIABETIC YEARS IN THE STUDY POPULATION

S.No	Duration of Diabetic years	With EDS	Without EDS
1	0-5	46	49
2	6-10	15	22
3	11-15	13	6
4	16-20	3	3
5	21-25	2	1

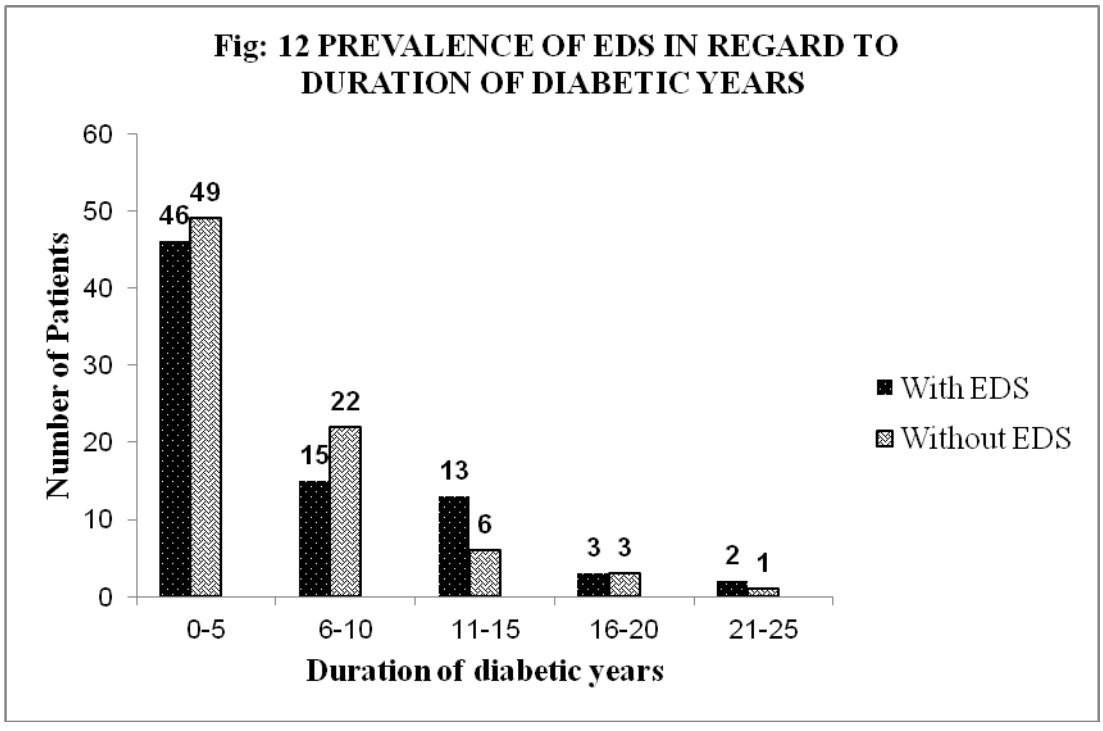


Table: 13 PERCENTAGE PREVALENCE OF EDS IN THE SUBJECTS WITH REGARD TO SOCIAL HABITS

S.No	Social Habits	With EDS(%)	Without EDS
1	Smoker	22.2	15.2
2	Alcoholic	11.1	7.6
3	Tea/Coffee	98.7	95

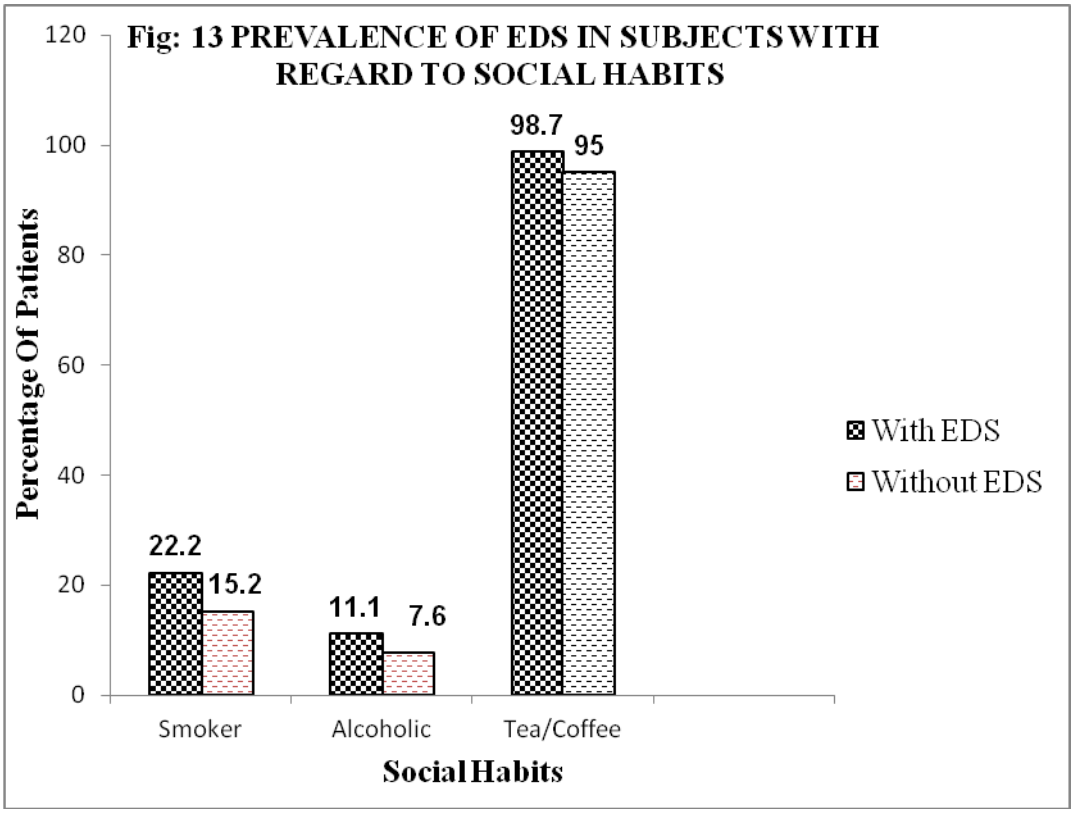


Table: 14 ASSOCIATION OF CO-MORBID DISEASE CONDITION IN DIABETIC PATIENTS WITH EDS

S.No	Co-morbid Disease condition	Percentage of Patients
1	Hypertension	36.7
2	Hypercholestremia	11.3
3	Foot Ulcer	27.8
4	Heart Disease	3.8
5	Respiratory Disease	5.1
6	Acidity	5.1
7	Blurred Vision	3.8
8	Kidney Disease	7.6

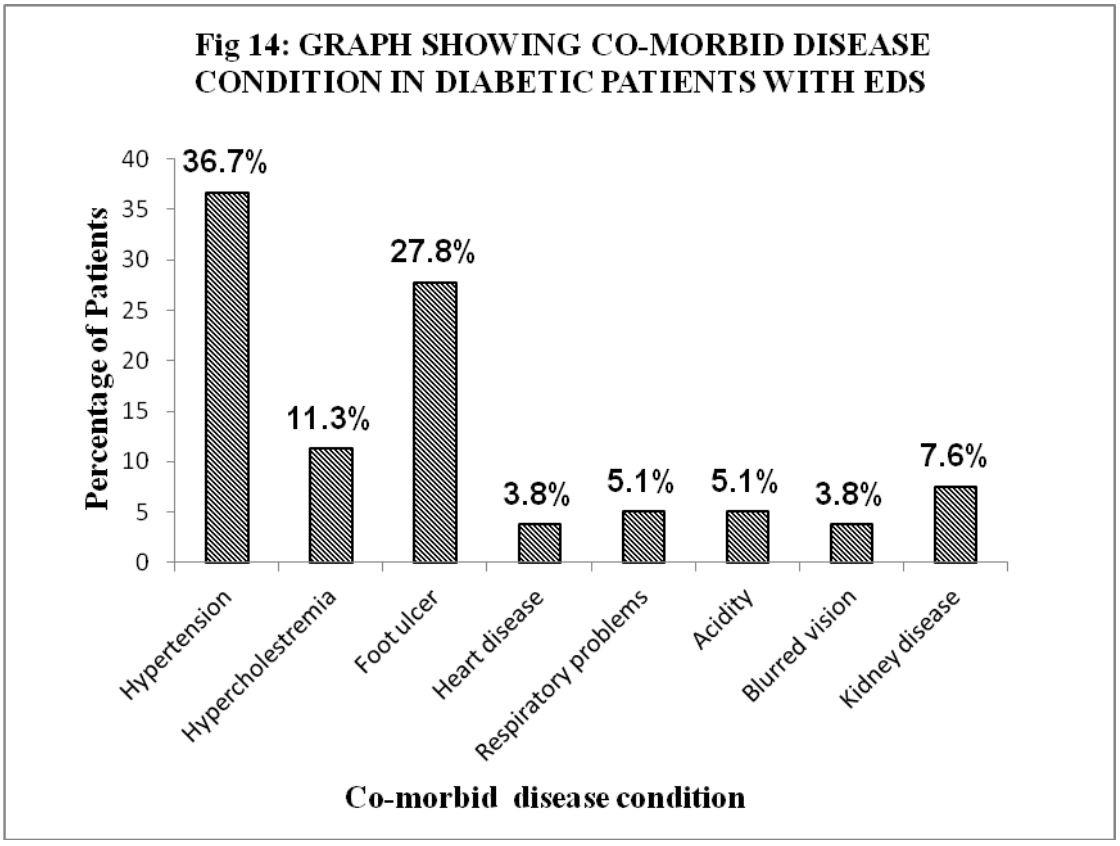


Table: 15 FASTING BLOOD SUGAR LEVEL IN DIABETIC PATIENTS WITH EDS

S.No	FBS	Percentage of Patients
1	Normal (60-100 mg/dl)	5.1
2	Abnormal (above 100 mg/dl)	94.9

Table: 16 POST PRANDIAL BLOOD SUGAR LEVEL IN DIABETIC PATIENTS WITH EDS

S.No	PPBS	Percentage of Patients
1	Normal (120-180 mg/dl)	8.9
2	Abnormal (above 180 mg/dl)	91.1

Table: 17 GLYCOSYLATED HAEMOGLOBIN LEVEL IN DIABETIC PATIENTS WITH EDS

S.No	HbA1C	Percentage of Patients
1	Normal (5.7-6.9%)	11.4
2	Abnormal(above 7)	88.6

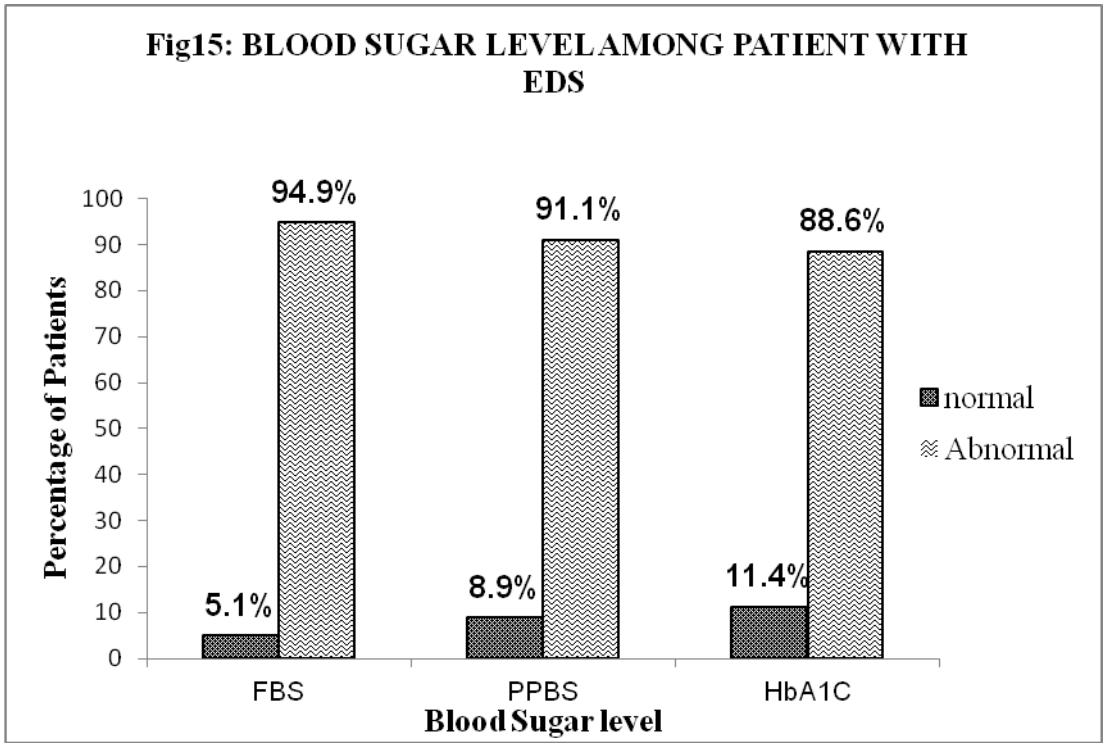


Table: 18 COMPARISON IN SLEEPING QUALITY USING EPWORTH SLEEPINESS SCALE ACCORDING TO GENDER

Variables	Male(N=40)	Female(N=39)
Sitting and reading		
Never fall asleep	**15(37.5%)**	**14(35.9%)**
Slight chance of falling asleep	**4(10%))**	**1(2.6%)**
Medium chance of falling asleep	**11(27.5%)**	**12(30.8%)**
High chance of falling asleep	**10(25%)**	**12(30.8%)**
Watching TV		
Never fall asleep	**3(7.5%)**	**0**
Slight chance of falling asleep	**10(25%)**	**12(30.8%)**
Medium chance of falling asleep	**15(37.5%)**	**13(33.3%)**
High chance of falling asleep	**12(30%)**	**14(35.9%)**
Sitting, inactive in a public place like a theatre or meeting		
Never fall asleep	**3(7.5%)**	**4(10.3%)**
Slight chance of falling asleep	**8(20%)**	**8(20.5%)**
Medium chance of falling asleep	**25(62.5%)**	**22(56.9%)**
High chance of falling asleep	**4(10%)**	**5(12.8%)**
As a passenger in a car for an hour without a break		
Never fall asleep	**0**	**0**
Slight chance of falling asleep	**6(15%)**	**2(5.2%)**
Medium chance of falling asleep	**4(10%)**	**7(17.9%)**
High chance of falling asleep	**30(75%)**	**30(76.9%)**
Lying down to rest in the afternoon when circumstances permit		
Never fall asleep	**2(5%)**	**0**
Slight chance of falling asleep	**0**	**0**
Medium chance of falling asleep	**1(2.5%)**	**1(2.5%)**
High chance of falling asleep	**37(92.5%)**	**38(97.4%)**
Sitting and talking to someone		
Never fall asleep	**30(75%)**	**30(76.9%)**
Slight chance of falling asleep	**6(15%)**	**6(15.4%)**
Medium chance of falling asleep	**1(2.5%)**	**0**
High chance of falling asleep	**3(7.5%)**	**3(7.7%)**

Variables	Male(N=40)	Female(N=39)
Sitting quietly after lunch without alcohol		
Never fall asleep	**3(7.5%)**	**1(2.6%)**
Slight chance of falling asleep	**35(87.5%)**	**33(84.6%)**
Medium chance of falling asleep	**1(2.5%)**	**4(10.3%)**
High chance of falling asleep	**1(2.5%)**	**1(2.6%)**
In a car, while stopped for a few minutes in the traffic		
Never fall asleep	**33(82.5%)**	**33(84.6%)**
Slight chance of falling asleep	**6(15%)**	**3(7.7%)**
Medium chance of falling asleep	**0**	**0**
High chance of falling asleep	**1(2.5%)**	**3(7.7%)**

Table: 19 SLEEPING QUALITY AND ITS PATTERNS IN THE DIABETIC POPULATION USING PITTSBURGH SLEEP QUALITY INDEX (PSQI) (N=160)

Variables	Patients with disturbed sleep (N=79)	Patients with good Sleep (N=81)
During the past month, how often have you taken medicine to help you sleep		
Never	**72(91.1%)**	**79(97.5%)**
Once a week	**2(2.5%)**	**1(1.2%)**
More than once a week	**5(6.3%)**	**1(1.2%)**
Wake up in the middle of the night or early morning		
Never	**2(2.5%)**	**10(12.3%)**
Once a week	**38(48.1%)**	**29(35.8%)**
More than once a week	**39(49.4%)**	**42(51.9%)**
Cannot get to sleep within 30 minutes		
Never	**20(25.3%)**	**49(60.5%)**
Once a week	**30(37.9%)**	**2024.7%)**
More than once a week	**29(36.7%)**	**12(14.8%)**
Have to get up to use the bathroom		
Never	**1(1.3%)**	**2(2.9%)**
Once a week	**4(5.1%)**	**12(14.8%)**
More than once a week	**74(93.7%)**	**67(82.7%)**
Had bad Dreams		
Never	**28(35.4%)**	**52(64.2%)**
Once a week	**32(40.5%)**	**28(34.6%)**
More than once a week	**19(24.1%)**	**1(1.2%)**
Have Pain		
Never	**24(30.4%)**	**56(69.1%)**
Once a week	**7(8.9%)**	**13(16%)**
More than once a week	**48(60.8%)**	**12(14.9%)**

RESULTS

The prospective study was aimed to find out the probable overall prevalence of excessive day time sleepiness in diabetic and obese patients. A total of 160 patients with Type II Diabetes Mellitus who were attending diabetic clinic at KMCH, Coimbatore were selected as the subjects for the study.

The study population consisted of 88 males and 72 females (Table 1, Fig 1) with an average age of 52.06± 8.822 and 50.55 ± 8.838 respectively.

The majority of patients among study population was within the age group of 56 to 65 nearly 38.7% followed by 46 to 55 with the percentage of 33.7% followed by 36 to 45 with the percentage of 22.5% & 25-35 with a percentage of 5%. (Table 2, figure 2).

The study populations were categorized based on their educational status. It was found that 12.5 % of males and 33.3 % of females were illiterates.25% of male and 20.5 % of female were having educational qualification up to 10 standard, 37.5 % of male and 23.1% of female were having qualification up to 12 standard, remaining 25% of male and 23.1% of female were having a qualification of degree or above (Table3, Fig: 3).The study population was also categorized based on their monthly income. 46.8% of the population were having a monthly income of less than 5000, 36.7% of population were having a monthly income between 5000 to 15,000 and the remaining 16.5% of population were having a monthly income of more than 15,000.(Table 4,Fig:4)

The prevalence of Excessive Day time Sleepiness in the study population was found by Epworth Sleepiness Scale and Pittsburgh Sleep Quality index. Among the study population, 40 males and 39 females were found to have EDS whereas 48 males and 33 females were found without EDS. (Table 5, Fig: 5).In the study population 45.4% of males

and 54.2% of females were found to have EDS (Table6, Fig:6.1&Fig:6.2). ESS score revealed that diabetic females were significantly sleepier than diabetic males during the day time. The majority of patients with EDS were within the age group of 56-65 (36), followed by 46-55 (25), followed by 36-45(14), and followed by 25-35 (4). (Table: 7, Fig: 7). The association of prevalence of EDS and age group was found out by using chi-square test ($p \geq 0.300$) indicating that age group is not significantly associated to EDS although older age group are more prone to disease.

Body mass index (BMI) was calculated. Based on the BMI study subjects were classified into three groups 18.5 to 24.9, 25 to 29.9 and ≥ 30. According to ESS Scale, patients were classified as with EDS and without EDS. In the group of BMI (18-24.9) 31.2% of patients were with EDS and 68.8 % of patients were without EDS. In the group of BMI(25-29.9), 62.3% of patients were with EDS and 37.7% of patients were without EDS. In the group of BMI ≥ 30 84.2% of patients were with EDS and 15.8% of patients were without EDS. It clearly indicates that BMI is significantly associated with EDS (p=0.000) (Table: 8,Fig: 8).Among the subjects with EDS, 37.5% of males and 25.6% of females were having BMI in the range of 18-24.9(normal),40% of males and 56.4% of females were having BMI in the range of 25-29.9 (overweight) and 22.5% of males and 17.9% of females were having a BMI ≥30 (obese).(Table:9,Fig: 9). It clearly indicates that obesity is higher in diabetic females who had high chances of EDS than males.

Based on the physical activity, study subjects were classified into two groups, one having moderate and other having sedentary physical activity. In the group having moderate physical activity 26 % were with EDS and 74% were without EDS. In the group leading a sedentary lifestyle 59.5% were with EDS and 40.5% were without EDS. It clearly indicates a strong association of EDS with physical activity (p=0.000)(Table:10,Fig:10). Among the

subjects with EDS, 77.5% of males and 89% of females were leading a sedentary life style and 22.5% of males and 10.25% of females were having moderate physical activity. (Table 11, Fig:11) It clearly indicates that physical activity is lower in diabetic females with poor sleep compared to males.

Concerned with the duration of diabetes in the study population, 46 patients with EDS and 49 patients without EDS had the disease for a period of 0-5 years; 15 patients with EDS and 22 patients without EDS had disease for 6-10 years; 13 patients with EDS and 6 patients without EDS had disease for 11-15 years; 3 patients with EDS and 2 patients without EDS had disease for 16-20 years; and 2 patients with EDS and 1 patient had disease for 21-25 years of experience.(Table:12,Fig:12) Related to the laboratory data 94.9 % had abnormal FBS and 5.1% had normal FBS values, 91.1% had abnormal PPBS and 8.9% had normal PPBS values, 88.6% had normal HbA1C and 11.4% had abnormal HbA1C.(Table15,16,17,Fig: 15)

Among the study population ,the subjects were asked about their social habits and it was found that 22.2 % of patients with EDS and 15.2% of patients without EDS were smokers; 11.1% of patients with EDS and 7.6% of patients without EDS were alcoholics & 98.2% of patients with EDS and 95% of patients without EDS have a habit of taking either tea /coffee.(Table 13,Fig:13) Shift workers are more prone to EDS. Out of 5 shift workers who visited Diabetic clinic during the study period ,4 were having EDS.

Risk factors like hypertension (36.7%), Foot Ulcer (27.8), Hypercholestremia (11.3%) and heart disease (3.8%) were more frequent in diabetic patients with EDS.(Table 14,Fig:14)

Table 19 shows the sleeping quality, excessive daytime sleepiness and its patterns in the diabetic population using the Pittsburgh sleep quality index (PSQI).72(91%) of the

patients with disturbed sleep and 79(97.5%) of the patients with good sleep have never taken any medicine to help them sleep. 2(2.5%) of the patients with disturbed sleep and 1 (1.2%) of the patients with good sleep have taken medicine once a week to help them sleep. 5(6.3%) of the patients with disturbed sleep 1(1.2%)of the patient with good sleep have taken medicine more than once in a week to help them sleep.2(2.5%) of the patients with disturbed sleep and 10 (12.3%) of the patients with good sleep have never waken up in the middle of the night or early morning.38(48.1%) of the patients with disturbed sleep and 29(35.8%) of the patients with good sleep have waken up once a week in the middle of the night or early morning.39(49.41%)of the patients with disturbed sleep and 42(51.9%) of the patients with good sleep have waken up more than once in a week.20(25.3%) of the patients with disturbed sleep and 49(60.5%) of the patients with good sleep have never taken more than 30 minutes to get sleep.30(37.9%) of the patients with disturbed sleep and 20(24.7%) of the patients with good sleep have taken more than 30 minutes to get sleep once a week.29(36.7%) of the patients with disturbed sleep and 12(14.8%) of the patients with good sleep have taken more than 30 minutes to get sleep more than once in a week.1(1.3%) patients with disturbed sleep and 2(2.9%) patients with good sleep have never got up to use the bathroom.4(5.1%) patients with disturbed sleep and 12(14.8%) patients with disturbed sleep have got up once a week to use the bathroom.74(93.7%) patients with disturbed sleep and 67(82.7%) patients with good sleep have got up more than once in a week.28(35.4%) with disturbed sleep and 52(64.2%) with good sleep never had bad dreams.32(40.5%) patients with disturbed sleep and 28(34.6%) of patients with good sleep had bad dreams once a week.19(24.1%) patients with disturbed sleep and 1(1.2%) patient with good sleep had bad dreams more than once in a week. 24(30.4%) of patients with disturbed sleep and 56(69%) patients with good sleep never had pain.7(8.9%) patients with disturbed sleep and 13(16%)

patients with good sleep had pain once a week.48(60.8%) patients with disturbed sleep and 12(14.9%) patients with good sleep had pain more than once in a week.

Table18 shows the comparison in sleeping quality in studied subjects using Epworth sleepiness scale (ESS). Female diabetic patients had high chances of falling asleep during the day time than males; sitting and reading (30.8% vs 25%), watching TV (35.9% vs 30%) sitting inactive in a public place (12.8% vs 10%), as a passenger in a car (76.9%% vs 75%), lying down to rest in the afternoon (97.4% vs 92.5%), sitting and talking to someone (7.7% vs 7.5%),sitting quietly after lunch without alcohol(2.6% vs 2.5%) and in a car while stopped for few minutes in traffic (7.7% vs 2.5%). ESS score results showed that diabetic women were sleepier (54.2 %) than men (45.4 %) during the day-time.

DISCUSSION

Diabetes mellitus is a major public health problem, causing significant morbidity and mortality. Across different populations, several studies have found sleep disturbances in patients with diabetes mellitus in relation with their quality of life. However, the relationship between sleep disorders and diabetes mellitus is less understood and less studied.The current study is a study using the Epworth sleepiness scale (ESS) and Pittsburgh sleep quality index (PSQI) to examine the sleep quality and excessive day-time sleepiness (EDS) in the diabetic population. Recent literature suggests that it may also be associated with the metabolic syndrome, for example: obesity, diabetes, insulin resistance. Even in the present study, the data revealed that there is a strong association between EDS and diabetes. This finding suggests that diabetes should be considered whenever a complaint of EDS is present in individuals.[71-73]

A study conducted by **Abdulbari Bener et al** has demonstrated a high proportion of sleep loss (60.1%) in Arab diabetic population residing in the State of Qatar. Among the studied diabetic patients, female diabetic patients (57%) were likely to have more sleep loss than male (43%) in the Arab population in Qatar.[36] It was also reported in a study by **Suarez** that Sleep quality and symptoms of poor sleep have been linked to increased risk of type 2 DM with recent evidence suggesting stronger associations in women.[74]

In the present study, diabetic patients (49.4%) reported high chances of daytime sleepiness. Among the studied diabetic patients, female diabetic patients (54.2 %) were likely to have excessive daytime sleepiness than males (45.4%). Another study done in Japan among male population, **Kawa-kami et al** reported a high incidence of diabetes in male subjects reporting sleeping disturbances after controlling other factors relevant to type 2 DM. These studies identify

sleep as a potential factor influencing glucose control in a specific population of patients with type 2 DM.[75]

In this study, quality of sleep varied by gender in diabetic patients. There was a considerable difference observed in ESS scores between both sexes. This is in contrast with another study by **Walker RD et al** that no significant difference was observed in ESS scores between both genders[76].

Female diabetic patients had high chances of falling asleep during the daytime than men especially while sitting and reading. (30.8% vs 25%), watching TV (35.9% vs 30%) sitting inactive in a public place (12.8% vs 10%), as a passenger in a car (76.9%% vs 75%), lying down to rest in the afternoon (97.4% vs 92.5%), sitting and talking to someone (7.7% vs 7.5%),sitting quietly after lunch without alcohol(2.6% vs 2.5%) and in a car while stopped for few minutes in traffic (7.7% vs 2.5%). Another study by **Meisinger et al** demonstrated that the men and women who reported a high frequency of sleep loss had a significantly higher risk for type 2 DM.[77]

Thus, poor diabetes control could contribute both to a higher perceived sleep debt and lower sleep quality. Sleep disturbances with DM could be caused by either physical or psychological discomfort due to the disorder. This shows that quality of life is vital for health and well being in persons with type 2 DM.

E.O Bixler et al in their study observed a strong association between EDS and BMI. Until the overweight threshold was reached, the BMI specific prevalence of EDS remained constant. Beyond this BMI threshold, the prevalence of EDS increased in an exponential manner.[40] This is in accordance with the present study. In the group of BMI (18-24.9) 31.2% of patients were with EDS and 68.8 % of patients were without EDS. In the group of BMI(25-

29.9), 62.3% of the patients were with EDS and 37.7% of the patients were without EDS. In the group of BMI ≥ 30 84.2% of the patients were with EDS and 15.8% of the patients were without EDS. **Abdulbari Bener et al** in their study found that obesity was significantly higher in diabetic females with higher chances of falling asleep during day time (51.7%)than in males(39.3%).[36] In the present study, among the subjects with EDS 40% of males and 56.4% of females were having BMI in the range of 25-29.9 (overweight) and 22.5% of males and 17.9% of females were having a BMI ≥30 (obese).Physical activity was significantly less in diabetic females(38.6%) compared to men (50.2%).[36]This result is also in accordance with the present study. In the group leading a sedentary lifestyle 59.5% were with EDS and 40.5% were without EDS. It clearly indicates a strong association of EDS with physical activity. Among the subjects with EDS, 77.5% of males and 89% of females were leading a sedentary life style.

Sleep efficiency declines with increasing age. With increasing age the homeostatic sleep mechanism weakens affecting both night time sleep efficiency and day time sleep propensity[40] .The majority of patients with EDS were within the age group of 56-65 (36), followed by 46-55 (25), followed by 36-45(14), and followed by 25-35 (4).

Foley D et al., in their study reported that that males and females with sleep disturbance were more likely to be obese to have a history of hypertension.[13] 36.7% of patients with EDS had hypertension. Hence the alarming rise in sleep loss among female population is due to rising problem of obesity and chronic diseases.

CONCLUSION

The study findings observed that excessive day time sleepiness is more prevalent in diabetic obese population. Sleep loss varies considerably by gender in diabetic patients. It was more observed in diabetic females than males. Obesity was also more common in diabetic women than in men while physical activity was less in women compared to men. A sedentary lifestyle has shown to enhance excessive day time sleepiness in diabetic patients. Difference was observed in the ESS scores between both genders. Sleep disturbances were seen more common in older age group of 55-65. Most of the patients observed with EDS had hypertension as a co-morbid disease.

Sleep exerts marked modulatory effects on glucose metabolism by influencing the balance and levels of hormones, including leptin, ghrelin, insulin and cortisol, in addition to the activity of the sympathetic nervous system(SNS). These physiological defence mechanisms alter glucose tolerance and sensitivity to insulin and leptin, impairing appetite regulation. Therefore, when chronically activated, these stress responses favour the development of obesity and T2DM. In addition, shorter sleepers have more opportunity for food consumption and tend to be more fatigued and less active during the day, which favour obesity. On the other hand, obesity itself, especially when central, leads to OSA, to inflammatory processes, and to the development of T2DM. Thus, we may conclude that it is difficult to isolate a cause and an effect from this neuro-endocrine-metabolic misbalance, since sleep characteristics (disorders or duration) may impact neurological and endocrine systems to promote obesity and T2DM, while obesity and T2DM may impact sleep as well. Therefore, while obesity and T2DM are favoured

and aggravated by short sleep duration or sleep disorders, sleep may be impaired by these two widespread metabolic conditions.

Thus sleep quality is seen poor in diabetic patients. To improve sleep quality, a clinical pharmacist can help patients to follow good sleep habits. The pharmacist encourages patients to engage in good sleep hygiene to reduce daytime sleepiness and instruct patients that adequate high-quality sleep is important to improve daytime function. Good sleep hygiene includes ensuring adequate sleep duration, developing sleep promoting bedtime rituals, avoiding staying in bed if unable to sleep, and avoiding caffeine if it disturbs the patient's ability to fall asleep. There are many effective treatments for sleep disorders, and the deleterious health effects of insufficient sleep or a coexisting sleep disorder warrant greater attention. Pharmacist can be instrumental in encouraging adherence to treatment for sleep disorders. Although persons with diabetes are instructed to restrict calories and to increase physical activity, the presence of less than optimal sleep may undermine these important treatment goals. Thus, educating patients with type 2 diabetes about the importance of sleep and regular screening for sleep disorders has the potential for a positive clinical outcome.

Thus the need of setting up of **Sleep Disorder** clinics at diabetological centres have become quiet an essential part, as the number of diabetic obese population is increasing at an alarming rate day by day. This enables the diabetic population to improve their quality of life.

BIBLIOGRAPHY

1) Andreoli T.E; 2003. *Dorland's illustrated medical dictionary* 30 th ed. Philadelphia Saunders Publications.

2) Group a. The American Academy of Sleep Medicine: The international Classification of Sleep disorders In Group, ed. Revised.Diagnostic and coding Manual.Rochester NY:Davies Printing.Co;1997

3) Pagel J.F. Excessive Day Time Sleepiness. American Family Physician.2009 Mar;79(5):391-396

4) Sicree R J,Shaw J,Zimmet P.The global burden,diabetes and impaired glucose tolerance.In: IDF diabetes atlas. 4th ed., Brussels: International Diabetes Federation.2009.1-105

5) Knutson KL,Ryden AM,Mander BA,Van Cauter E.Role of Sleep duration and quality in the risk and severity of type 2 diabetic mellitus.Arch Intern Med 2006;166:1768-1774

6) Trento M,Broglio F,Riganti F,Basile M, Borgo E,Kucich C,et al.Sleep abnormalities in type 2 diabetes may be associated with glycemic control.Acta Diabetol 2008; 45:225-229

7) West SD, Nicoll DJ, Stradling JR. Prevalence of obstructive sleep apnea in men with type 2 diabetes. Thorax 2006; 61:945–950.

8) Villa MP, Multari G, Montesano M, Pagani J, Cervoni M, Midulla F, et al. Sleep apnea in children with diabetes mellitus: effect of glycemic control. Diabetologia 2000;43:696–702.

9) James PT. Obesity: the worldwide epidemic. Clin Dermatol 2004;22:276–280.

10) Chaput JP, Despre´s JP, Bouchard C, Astrup A, Tremblay A. Sleep duration as a risk factor for the development of type 2diabetes or impaired glucose tolerance: analyses of the Quebec Family Study. Sleep Med 2009;10:919–924.

11) Van Cauter E, Splegel K, Tasali E, Leproult R. Metabolic consequences of sleep and sleep loss. Sleep Med 2008;9:S23–28

12) Pillar G, Shehadeh N. Abdominal fat and sleep apnea: the chicken or the egg? Diabetes Care 2008;31(Suppl. 2):S303–309

13) Foley D, Ancoli-Israel S, Britz P, Walsh J, Sleep disturbances and chronic disease in older adults: results of the 2003 National Sleep Foundation Sleep in America Survey. J Psychosom Res 2004; 56: 497-502

14) Bonnet MH, Arand DL, We are chronically sleep deprived. Sleep, 1995; 18: 908-911

15) Resnick HE, Redline S, Shahar E et al, Diabetes and Sleep disturbances: Findings from the Sleep Heart Study. Diabetes Care; 2003; 26: 702-709

16) Leproult R, Holmback U, Van Cauter E, Marked decrease in insulin sensitivity following one week of partial sleep deprivation with or without circadian mis-alignment. Diabetes 2006; 55: A323-A324

17) Spiegel K, Leproult R, Van Cauter E, Impact of Sleep debt on metabolic and endocrine function. Lancet 1999; 354: 1435-1439

18) Excessive day time sleepiness [online]. Available from-Wikipedia, the free encyclopedia.http://en.wikipedia.org/wiki/Excessive_daytime_sleepiness.

19) Barone M.T.U, Menna-Barreto. L,Diabetes and Sleep: A complex cause- and- effect relationship. Diabetes Research and Clinical Practice 2011;91:129-137

20) Spiegel K, Knutson K, Leproult R, Tasali E, Van Cauter E. Sleep loss: a novel risk factor for insulin resistance and type 2 diabetes. J Applied Physiology 2005;99:2008–2019.

21) Mallon L, Broman JE, Hetta J. High incidence of diabetes inmen with sleep complaints or short sleep duration: a 12-year follow-up study of a middle-aged population. Diabetes Care 2005; 28:2762–2767.

22) Gonzalez-Ortiz M, Martinez-Abundis E, Balcazar-Munoz BR, Pascoe-Gonzalez S. Effect of sleep deprivation on insulin sensitivity and cortisol concentration in healthy subjects. Diabetes Nutr Metab 2000;13:80–83.

23) Gangwisch JE, Heymsfield SB, Boden-Albala B, Bujis RM, Kreier F, Pickering TG, et al. Sleep duration as a risk factor for diabetes incidence in a large US sample. Sleep2007; 30:1667–1673.

24) Nakajima H, Kaneita Y, Yokoyama E, Harano S, Tamaki T, Ibuka E, et al. Association between sleep duration and hemoglobin A1c level. Sleep Med 2008; 9:745–752.

25) Yaggi HK, Araujo AB, McKinlay JB. Sleep duration as a risk factor for the development of type 2 diabetes. Diabetes Care 2006; 29:657–661.

26) Gronfier C & Brandenberger G. Ultradian rhythms in pituitary and adrenal hormones: their relations to sleep. Sleep MedRev 1998; 2: 17–29

27) Steiger A. Sleep and endocrinology. J Intern Med 2003; 254: 13–22.

28) Van Cauter E. Endocrine physiology. In Kryger M, Roth T & Dement WC (eds.). Principles and practice of sleep medicine. 4th ed. Philadelphia: Elsevier-Saunders, 2005, pp. 266–282.

29) Sakurai T. The neural circuit of orexin (hypocretin): maintaining sleep and wakefulness. Nat Rev Neurosci 2007; 8: 171–181.

30) De Lecea L & Sutcliffe JG. The hypocretins and sleep. FEBS J 2005; 272: 5675 - 5688.

31) Vgontzas AN, Papanicolaou DA, Bixler EO, Kales A,Tyson K,Chrousos GP.Elevation of plasma cytokines in disorders of excessive daytime sleepiness: Role of sleep disturbance and obesity. Journal of Clinical Endocrinology and Metabolism 1997;82(5):1313-1316

32) Pickup JC. Inflammation and activated innate immunity in the pathogenesis of type 2 diabetes.Diabetes Care 2004; 27:813-823

33) Dixon JB, Dixon ME, Anderson ML, Schachter L, O' Brien PE, Daytime sleepiness in the obese: Not as simple as obstructive sleep apnea.Obesity.2007;15(10):2504-2511.

34) Johns MW. Daytime sleepiness, snoring, obstructive sleep apnea -the Epworth Scale. Chest 1993;103:30-36

35) Johns MW.A new method for measuring daytime sleepiness: the Epworth sleepiness scale.Sleep 1991;14:540-545

36) Bener.A, Al-Hamaq A.O.A.A, Sleep quality and excessive daytime sleepiness in a Arab diabetic population. Biomedical Research 2010;22(1): 333-340.

37) Adriana R, Prevalence and predictors of excessive day time sleepiness in Romanian obese type 2 diabetic patients. Applied Medical Informatics.2011

38) Resto O. Foschino- Barbaro MP, LeqariG, Talamo S,Bonfitto P,Palumbo A,Minenn A,Giorgino R,De Pergola G,Sleep related breathing disorders,loud snoring and EDS in obese subjects.International journal journal of obesity and related metabolic disorders.2001 May 25(5): 669-675

39) Ruggles K, Hausman N, Evaluation of excessive day time sleepiness. Wisconsin medical journal 2003;102(1):21-24.

40) Bixler E.O, Vgontzas A.N, Lin H.-M, Calhoin S.L, Vela-Bueno A and Kales A, Excessive daytime sleepiness in a general population sample: The role of Sleep apnea, age, obesity, diabetes and depression. The journal of Clinical Endocrinology& Metabolism 2005;90(8): 4510-4515

41) Renko A-K, Hiltunen L, Laakso M, Rajala U, Keinanen-Kiukaanniemi S,The relationship of glucose tolerance to sleep disorders and daytime sleepiness.Diabetes Research and clinical Practice 2005;67:84-91

42) Chasens E.R, Umlauf M.G, Weaver T.E, Sleepiness, physical activity and functional outcomes in veterans with type 2 diabetes.Applied Nursing Research 2009;22:176-182

43) Tan W.C, Tze P N, Prevalence and determinants of excessive daytime sleepiness in an Asian multi-ethnic population. Sleep medicine 2005;6:523-529

44) Barone M.T.U, Barreto L.M, Diabetes and Sleep: A complex cause-and- effect relationship. Diabetes Research and Clinical Practice 2011;91:129-137

45) Banerjee D, Vitiello M.V, Grunstein R.R, Pharmacotherapy for excessive daytime sleepiness .Sleep medicine reviews 2004;8:339-354

46) American Academy of Sleep Medicine. *The International Classification of Sleep Disorders: Diagnostic & Coding Manual*. 2nd ed. Westchester,Ill.: American Academy of Sleep Medicine; 2005.

47) Pagel JF. Sleep disorders in primary care: evidence-based clinical practice.In: Pagel JF,Pandi-Perumal SR, eds. *Primary Care Sleep Medicine: A Practical Guide*. Totowa, N.J.: Humana Press; 2007:1-14.

48) Pagel JF. Medications that induce sleepiness. In: Lee-Chiong TL, ed. *Sleep:A Comprehensive Handbook*. Hoboken, N.J.: Wiley-Liss; 2006:175-182

49) Shaw JE, Punjabi NM, Wilding JP, Alberti KGMM, Zimmet PZ .Sleep-disordered breathing and type 2 diabetes—a report from the International Diabetes Federation Taskforce on Epidemiology and Prevention. Diabetes Research Clinical Practice 2008;81:2–12.

50) Punjabi NM, Polotsky VY. Disorders of glucose metabolism in sleep apnea. Journal of Applied Physiology 2005;99: 1998–2007.

51) Leproult R, Copinschi G, Buxton O, VanCauter E. Sleep loss results in an elevation of cortisol levels the next evening.Sleep 1997;20:865–870.

52) Straznicky NE, Eikelis N, Lambert EA, Esler MD. Mediators of sympathetic activation in metabolic syndrome obesity. Curr Hypertens Rep 2008;10:440–447.

53) Dijk DJ. Slow-wave sleep, diabetes, and the sympathetic nervous system. Proc Natl Acad Sci USA 2008;105:1107–1108.

54) Sandoval DA, Davis SN. Leptin – metabolic control and regulation. J Diabetes Complications 2003;17:108–113.

55) Balbo M, Leproult R, Van Cauter E. Impact of sleep and its disturbances on hypothalamo–pituitary–adrenal axis activity. International Journal of Endocrinology 2010;2010:1–16.

56) Qi D, Rodrigues B. Glucocorticoids produce whole body insulin resistance with changes in cardiac metabolism. Am J Physiol Endocrinol Metab 2007;292:E654–67.

57) Roberge C, Carpentier AC, Langlois MF, Baillargeon JP, Ardilouze JL, Maheux P, et al. Adrenocortical dysregulation as a major player in insulin resistance and onset of obesity.Am J Physiol Endocrinol Metab 2007;293:E1465–1478

58) Buckley TM, Schatzberg AF. Review: on the interactions of the hypothalamic–pituitary–adrenal (HPA) axis and sleep: normal HPA axis activity and circadian rhythm, exemplary sleep disorders. J Clin Endocrinol Metab 2005;90:3106–14.

59) Gale SM, Castracane VD, Mantzoros CS. Energy homeostasis, obesity and eating disorders: recent advances in endocrinology. J Nutr 2004;134:295–298

60) Martin SS, Qasim A, Reilly MP. Leptin resistance – a possible interface of inflammation and metabolism in obesity related cardiovascular disease. J Am Coll Cardiol 2008;52:1201–1210

61) Chen K, Li FH, Li J, Cai H, Strom S, Bisello A, et al. Induction of leptin resistance through direct interaction of C-reactive protein with leptin. Nat Med 2006;12:425–432.

62) Ip MSM, Lam KSL, Ho CM, Tsang KWT, Lam WK. Serum leptin and vascular risk factors in obstructive sleep apnea. Chest 2000;118:580–586.

63) Gil-Campos M, Aguilera CM, Canete R, Gil A. Ghrelin: a hormone regulating food intake and energy homeostasis. Br J Nutr 2006;96:201–226.

64) Knutson KL, Van Cauter E. Associations between sleep loss and increased risk of obesity and diabetes. Ann N Y Acad Sci 2008;1129:287–304.

65) Spiegel K, Leproult R, L'Hermite-Baleriaux M, Copinschi G, Penev PD, Van Cauter E. Leptin levels are dependent on sleep duration: relationships with sympathovagal balance, carbohydrate regulation, cortisol, and thyrotropin. J Clin Endocrinol Metab 2004;89:5762–5771.

66) Bastard JP, Maachi M, Lagathu C, Kim MJ, Caron M, Vidal H,et al. Recent advances in the relationship between obesity,inflammation, and insulin resistance. Eur Cytokine Netw2006;17:4–12

67) Kapsimalis F, Richardson G, Opp MR, Kryger M. Cytokines and normal sleep. Curr Opin Pulm Med 2005;11:481–484.

68) McClellan KJ, Spencer CM. Modafinil: a review of its pharmacology and clinical efficacy in the management of narcolepsy. *CNS Drugs*. 1998; 9(4):311-324.

69) Chervin RD. Sleepiness, fatigue, tiredness, and lack of energy in obstructive sleep apnea. *Chest*. 2000;118(2):372-379.

70) Czeisler CA, Walsh JK, Roth T, et al. Modafinil for excessive sleepiness associated with shift-work sleep disorder [published correction appears in *N Engl J Med*. 2005;353(10):1078]. *N Engl J Med*. 2005;353(5):476-486.

71) Wander PE, Brosson B, Aberg H, Quality of life in relation to co-morbidity among DM patients followed for 3 years in Swedish Primary Health Care, Diabetes Metab, 1999;25:424

72) Knutson KL, Spiegel K, Penev P, Van Cauter E, The metabolic consequences of sleep derivation, Sleep Med Rev, 2007;11:163-178

73) Martikainen K, Urponen H, Partinen M, Hassan J, Vuori I, Daytime Sleepiness : a risk factor in Community life, Acta Neurol Scand, 1992;86:337-341.

74) Suarez EC, Self reported symptoms of sleep disturbance and inflammation, coagulation, insulin resistance and psychosocial distress : Evidence for gender disparity, Brain Behav Immun, 2008;22:960-968

75) Kawakami N, Takatsuka N, Schimizu H : Sleep disturbance and onset of type 2 diabetes, Diabetes Care, 2004;27:282-283

76) Walker RD, Durazo-Arvizu R, Wachter B, Gopalsami C, Prospective difference between male and female patients with sleep apnea, Laryngoscope, 2001;111:1501-1505

77) Meisinger C, Heier M, Loewel H: MONICA/KORA Augsburg Cohort study Sleep disturbances as a predictor of type 2 DM in men and women from the general population, Diabetologia, 2005;48:235-241

Annexures

KMCH ETHICS COMMITTEE

KOVAI MEDICAL CENTER AND HOSPITAL LIMITED

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Ref: EC/AP/153/09/2011
19.09.2011

To

Ms. Josmy Jose,
M.Pharm II Year,
KMCH College of Pharmacy,
Coimbatore - 641 014

Dear Ms. Josmy Jose,

The proposal entitled "**A PROSPECTIVE STUDY ON PREVALENCE AND ASSESSMENT OF EXCESSIVE DAY TIME SLEEPINESS IN DIABETES AND OBESE POPULATION**" was reviewed by the Ethics Committee in its meeting held on 17.09.2011 and permission is granted to you to carryout the study at Kovai Medical Center and Hospital Ltd, Coimbatore, India.

Thanking you,

Yours faithfully,

Dr. P. R. Muthuswamy
Chairman, Ethics Committee

Dr. P. R. MUTHUSWAMY,
MA.,MBA.,FDPM(IIM-A)Ph.D.,
Chairman
Ethics Committee
Kovai Medical Center and Hospital
Avanashi Road,
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Epworth Sleepiness Scale

Name: _____

Date: _____

Your age: (Yr) _____ **Your sex**: ❑ Male ❑ Female

How likely are you to doze off or fall asleep in the situations described below, in contrast to feeling just tired?

This refers to your usual way of life in recent times.

Even if you haven't done some of these things recently try to work out how they would have affected you.

Use the following scale to choose the most appropriate number for each situation:-

0 = would never doze
1 = Slight chance of dozing
2 = Moderate chance of dozing
3 = High chance of dozing

Situation	**Chance of dozing**
Sitting and reading	
Watching TV	
Sitting, inactive in a public place (e.g. a theatre or a meeting)	
As a passenger in a car for an hour without a break	
Lying down to rest in the afternoon when circumstances permit	
Sitting and talking to someone	
Sitting quietly after a lunch without alcohol	
In a car, while stopped for a few minutes in the traffic	
Total	

Score:	
0-10	Normal range
10-12	Borderline
12-24	Abnormal

எவ்வொர்த்தின் பகல் தூக்கம் பற்றி அறிய உதவும் அளவுகோள்

பகல் நேரத்தில் தூங்கும் பழக்கம் இருக்கிறதா என்பதை கீழே கொடுக்கப்பட்டுள்ள கேள்வி பதில் மூலம் அறிந்து கொள்ளலாம்

0 - ஒருபோதும் தூக்கம் வருவதில்லை

1 - எப்போதாவது தூக்கம் வருதல்

2 - அடிக்கடி தூக்கம் வருதல்

3 - மிக அதிகமாக மயக்கமும் தூக்கமும் வருதல்

சூழ்நிலைக் காரணங்கள்

1. உட்கார்ந்து படிக்கும்போது
2. தொலைக்காட்சி அல்லது டிவி பார்க்கும்போது
3. பொது இடத்தில் அமர்ந்திருக்கும்போது அல்லது திரையரங்குகளில் இருக்கும்போது
4. ஒரு மணி நேரம் தொடர்ந்து வாகனத்தில் பயணம் செய்யும்போது
5. மதிய நேரத்தில் ஓய்வு எடுக்கும்போது
6. மற்றவர்களுடன் உட்கார்ந்து பேசும்போது
7. மதியம் சாப்பாட்டிற்குப் பிறகு அமர்ந்திருக்கும்போது (மது அருந்தாமல்)
8. காரில் பயணம் செய்யும்போது போக்குவரத்து நெரிசலில் உறக்கம் வருமா
9. மொத்த மதிப்பெண் :
10. நோயாளியின் பெயர் :
11. வயது :

12. முகவரி :

13. பால் : ஆண் / பெண்

14. நகரம் :

15. மாநிலம் :

16. பின்கோடு நம்பர் :

17. பிறந்த தேதி :

18. நாள் :

19. மருத்துவர் பெயர் :

கையொப்பம் மருந்தாளுநர் கையொப்பம்

Name:_____ Date: _____

Pittsburgh Sleep Quality Index (PSQI)

Instructions: The following questions relate to your usual sleep habits during the past month only. Your answers should indicate the most accurate reply for the majority of days and nights in the past month. **Please answer all questions.**

1. During the past month, what time have you usually gone to bed at night? _____
2. During the past month, how long (in minutes) has it usually taken you to fall asleep each night? _____
3. During the past month, what time have you usually gotten up in the morning? _____
4. During the past month, how many hours of actual sleep did you get at night? (This may be different than the number of hours you spent in bed.) _____

5. During the past month, how often have you had trouble sleeping because you…	Not during the past month	Less than once a week	Once or twice a week	Three or more times a week
a. Cannot get to sleep within 30 minutes				
b. Wake up in the middle of the night or early morning				
c. Have to get up to use the bathroom				
d. Cannot breathe comfortably				
e. Cough or snore loudly				
f. Feel too cold				
g. Feel too hot				
h. Have bad dreams				
i. Have pain				
j. Other reason(s), please describe:				
6. During the past month, how often have you taken medicine to help you sleep (prescribed or "over the counter")?				
7. During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?				
	No problem at all	Only a very slight problem	Somewhat of a problem	A very big problem
8. During the past month, how much of a problem has it been for you to keep up enough enthusiasm to get things done?				
	Very good	Fairly good	Fairly bad	Very bad
9. During the past month, how would you rate your sleep quality overall?				

	No bed partner or room mate	Partner/room mate in other room	Partner in same room but not same bed	Partner in same bed
10. Do you have a bed partner or room mate?				
	Not during the past month	Less than once a week	Once or twice a week	Three or more times a week
If you have a room mate or bed partner, ask him/her how often in the past month you have had:				
a. Loud snoring				
b. Long pauses between breaths while asleep				
c. Legs twitching or jerking while you sleep				
d. Episodes of disorientation or confusion during sleep				
e. Other restlessness while you sleep, please describe:				

Scoring the PSQI

The order of the PSQI items has been modified from the original order in order to fit the first 9 items (which are the only items that contribute to the total score) on a single page. Item 10, which is the second page of the scale, does not contribute to the PSQI score.

In scoring the PSQI, seven component scores are derived, each scored 0 (no difficulty) to 3 (severe difficulty). The component scores are summed to produce a global score (range 0 to 21). Higher scores indicate worse sleep quality.

Component 1: Subjective sleep quality—question 9

Response to Q9	Component 1 score
Very good	0
Fairly good	1
Fairly bad	2
Very bad	3

Component 1 score:_____

Component 2: Sleep latency—questions 2 and 5a

Response to Q2	Component 2/Q2 subscore
≤ 15 minutes	0
16-30 minutes	1
31-60 minutes	2
> 60 minutes	3

Response to Q5a	Component 2/Q5a subscore
Not during past month	0
Less than once a week	1
Once or twice a week	2
Three or more times a week	3

Sum of Q2 and Q5a subscores	Component 2 score
0	0
1-2	1
3-4	2
5-6	3

Component 2 score:_____

Component 3: Sleep duration—question 4

Response to Q4	Component 3 score
> 7 hours	0
6-7 hours	1
5-6 hours	2
< 5 hours	3

Component 3 score:_____

Component 4: Sleep efficiency—questions 1, 3, and 4

Sleep efficiency = (# hours slept/# hours in bed) X 100%

hours slept—question 4

hours in bed—calculated from responses to questions 1 and 3

Sleep efficiency	Component 4 score
> 85%	0
75-84%	1
65-74%	2
< 65%	3

Component 4 score:_____

Component 5: Sleep disturbance—questions 5b-5j

Questions 5b to 5j should be scored as follows:

Not during past month	0
Less than once a week	1
Once or twice a week	2
Three or more times a week	3

Sum of 5b to 5j scores	Component 5 score
0	0
1-9	1
10-18	2
19-27	3

Component 5 score:_____

Component 6: Use of sleep medication—question 6

Response to Q6	Component 6 score
Not during past month	0
Less than once a week	1
Once or twice a week	2
Three or more times a week	3

Component 6 score:_____

Component 7: Daytime dysfunction—questions 7 and 8

Response to Q7	Component 7/Q7 subscore
Not during past month	0
Less than once a week	1
Once or twice a week	2
Three or more times a week	3

Response to Q8	Component 7/Q8 subscore
No problem at all	0
Only a very slight problem	1
Somewhat of a problem	2
A very big problem	3

Sum of Q7 and Q8 subscores	Component 7 score
0	0
1-2	1
3-4	2
5-6	3

Component 7 score:_____

Global PSQI Score: Sum of seven component scores:_____

Citation: Buysse, DJ, Reynolds CF, Monk TH, Berman SR, Kupfer DJ: The Pittsburgh Sleep Quality Index (PSQI): A new instrument for psychiatric research and practice. Psychiatry Research 28:193-213, 1989

பிட்ஸ்பர்க்கின் தூக்கத்தின் அளவு அறிய உதவும் அளவுகோல்

கீழே கொடுக்கப்பட்டுள்ள கடந்த மாதத்தில் உங்களுடைய தூக்கத்தின் அளவை பற்றியும், அளவுக்கு அதிகமான தூக்கத்தினால் ஏற்படும் பிரச்சனைகளைப் பற்றியும் அறிவு உதவும் அளவுகோல்.

பிட்ஸ்பர்க் சிலீப் (தூக்கம்) குவாலிட்டி ஸ்கேல்

வழிமுறைகள்

கீழே கொடுக்கப்பட்டுள்ள வினாக்கள் கடந்த ஒரு மாதத்தில் உங்களுடைய தூக்கத்தைப் பற்றி அறிய உதவுகிறது. உங்களுடைய பதில்கள் மிக சரியாக இருக்க வேண்டும். தயவு செய்து எல்லா கேள்விகளுக்கும் பதில் அளிக்கவும்.

1. **கடந்த ஒரு மாதத்தில் எத்தனை மணிக்கு நீங்கள் இரவில் தூங்க செல்வீர்கள்.**
2. **கடந்த மாதத்தில் எத்தனை முறை படுத்தவுடன் தூங்கியிருக்கிறீர்கள். எத்தனை நிமிடத்தில் தூங்கிவிடுவீர்கள்.**
3. கடந்த மாதத்தில் எத்தனை மணிக்கு காலையில் எழுவீர்கள் (அ) விழிப்பீர்கள்
4. கடந்த மாதத்தில் ஒரு நாளில் எவ்வளவு நேரம் இரவில் தூக்கத்தில் இருப்பீர்கள்? (இரு உங்களின் படுக்கையில் படுத்த நேரத்தைவிட தூங்கிய நேரம் மட்டும் குறிப்பிட வேண்டும்)

5. கடந்த ஒரு மாதத்தில் எத்தனை முறை உங்களுக்கு கீழே கொடுக்கப்பட்டுள்ள காரணத்தால் தூக்கத்தில் பிரச்சனைகளோ அல்லது பாதிப்போ ஏற்பட்டது.	கடந்த மாதத்தில் இல்லை	ஒரு வாரத்திற்கு குறைவாக	குறைந்தது ஒன்று அல்லது இரண்டு வாரத்தில்	மூன்று அல்லது அதற்கு மேலான வாரங்களில்

அ) படுத்த 30 நிமிடத்தில் தூக்கம் வரவில்லை				
ஆ) நடு இரவில் அல்லது விடியற்காலையில்				
இ) இரவில் பாத்ரூம் போக எழுந்திருக்கிறீர்களா?				
ஈ) இரவில் சரியாக சுவாசிக்க முடிகிறதா?				
உ) இரவில் இருமல் மற்றும் குறட்டை வருகிறதா?				
ஊ) இரவில் உடல் குளிர்வதுபோல் உணர்வீர்களா?				
எ) இரவில் உடல் வெப்பமாவது போல் உணர்கிறீர்களா?				
ஏ) இரவில் கெட்ட கனவுகள் வருகிறதா?				
ஐ) இரவில் உடலில் வலி இருக்கிறதா?				
ஒ) வேறு ஏதேனும் காரணங்கள் இருந்தால் விளக்கமாக சொல்லவும்				
6. கடந்த ஒரு மாதத்தில் எத்தனை முறை மாத்திரை எடுத்துக்கொண்டு தூங்கியிருக்கிறீர்கள் (மருத்துவரின் ஆலோசனையின் படி அல்லது நீங்களாகவா)				
7. கடந்த ஒரு மாதத்தில் வாகனம் ஓட்டும் போதோ சாப்பிடும்போதோ அல்லது பொது இடத்தில் செல்லும் போதோ எத்தனை முறை தூக்கம் வருவதுபோல் உணர்வீர்கள்?				

	ஒரு முறையும் தூக்கம் வரவில்லை	ஒரு தடவ மட்டும் தூக்கம் குறைவாக வந்து இருக்கு	ஒரு மாதிரி பிரச்சனை இருக்கு	அதிக முறை தூக்கம் இருக்கும்
8. கடந்த மாதத்தில் உங்கள் நடைமுறை வாழ்க்கையில் அன்றாட பணிகளை உற்சாகமாக செய்தீர்களா?				
	நல்ல முறையில்	ஒருவித முறையில்	ஒருவித மோசமான	அதிகமுறை மோசமானவை
9. கடந்த மாதத்தில் உங்களுடைய தூக்கத்தை நீங்கள் எப்படி உணர்கிறீர்கள்				
	படுக்கை துணை அல்லது ரூம் நண்பர் இல்லை	துணையோ அல்லது ரூம் நண்பர் வேறு எங்கேனும் இருக்கி-றார்களா	துணை உண்டு ஆனால் படுக்கை அறையில் இல்லை	படுக்கை அறையில் துணை உண்
10. உங்களுடன் ரூமில் நண்பர்கள் இருக்கிறார்களா? கடந்த ஒரு மாதத்தில் எத்தனை தடவை கீழே கொடுக்கப்பட்டுள்ள ஏதாவது அறிகுறிகள் உள்ளதா என்று அவர்களோடு கேட்டு தெரிந்து கொள்ளுங்கள்.				
அ) தூக்கத்தில் குறட்டைவிடுதல்				
ஆ) நீண்ட நேரம் தூங்கும் போது சுவாசம் விடுவதில் சிரமம் ஏற்படுகிறதா?				
இ) தூங்கும்போது காலை மடக்கவோ அல்லது நீட்டவோ செய்வதுண்டா?				
ஈ) தூங்கும்போது குழப்பமான மனநிலை போல் உணர்கிறீர்களா?				
உ) மற்ற ஏதாவது பிரச்சனைகள் இருந்தால் விளக்கமாக கூறுங்கள்?				

DATA ENTRY FORM

OP / IP No: ……	**Age: ….....Years Sex: Male / Female** **Nationality:**
Name: **Address:**	**Monthly Income: Rs: ……** **Education level : …….** **Occupation : …….**

Duration of Diabetes : …… Marital Status : ……. Physical activity:……

Height : ……. Weight : …… BMI : ……. Waist circumference : ……..

Smoking:…… Alcohol:…….. Tea/ Coffee: …….. Shift work: ……

RBS : ……mg/dL **Total cholesterol : ……. mg/dl**

FBS : ……mg/dL **HDL : ……..... mg/dl**

PPBS : …….....mg/dL **VLDL : ….…….. mg/dl**

HbA_1c : ……% **LDL : ...……mg/dl**

Blood Pressure : ……mm Hg **Triglycerides : ……....mg/dl**

Urine Analysis :

WHY DO WE NEED SLEEP?

- Rest and recharge our bodies
- Aid memory and learning
- Ensure normal hormone secretion
- Maintain normal immune function
- Heal and repair body tissues

- Maintain optimal emotional and social functioning while awake

TIPS ON GETTING TO SLEEP

- Aim for at least 6 to 8hours of uninterrupted sleep every night.
- Try to go to bed at the same time and get up at the same time every day - routine will encourage good quality sleep.
- Avoid naps during the day to ensure that you are tired at bedtime.
- Try to go to bed only when you are sleepy - this will reduce the amount of time you are awake in bed.

TIPS ON GETTING TO SLEEP cont…

- Try not to become stressed if you feel you are not getting enough sleep. Remember that sleep will come eventually, and try to relax.
- Avoid looking at the clock while in bed. If you can't get to sleep or get back to sleep for an extended period, get out of bed and do something boring in very dim light, or sit and relax in the dark until you are sleepy.
- Exercising in the morning or early afternoon can encourage a healthy sleep routine. Exercise 3 to 5times a week is recommended.
- Keep your bedroom at a cool, comfortable temperature, and maintain a dark and quiet sleeping environment.
- It's best not to watch television in bed, because this may stimulate your brain and delay sleep onset.

- Do not go to bed hungry similarly don't eat a heavy meal before bed. Both can interfere with sleep.
- Avoid consuming alcohol at least 4 hours before going to bed. It can seem to help you fall asleep, but it also causes disrupted sleep.
- Caffeine and tobacco are both stimulants; Avoid caffeine and smoking at least 4 hours before going to bed.